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RECURRENT VOMITING IN CHILDREN

ITS ETIOLOGY AND TREATMENT

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IN THIS country, children with recurrent attacks of vomiting, often known as cyclic vomiting, are seen more often in private practice than in hospital or out-patient work.* In this respect it is like a number of other diseases which occur in different strata of society. In England these attacks are said to be much more common in the dispensary class. Garrod's textbook[1] states that one-third of the patients seen in out-patient work are subject to it. Tallerman[2] collected 40 cases in a few months in the London Hospital. On the other hand, only 15 cases were diagnosed in the wards of the Children's Medical Service of Bellevue Hospital in twenty-five years, and cases are rarely seen in the out-patient department. It is a little difficult to reconcile these facts, and the question of different standards for diagnosis cannot be avoided. These cases are not very uncommon in private practice, however. Investigation, in the usual sense of the word, is somewhat more difficult with private patients, but, on the other hand, they are often followed for a long time, and information can be collected by continued observation which can be duplicated only with great difficulty in hospital practice. This paper is based on 65 cases seen in private and hospital work in the past twenty years with the addition of a few typical histories from the staff and from medical students (Table I). Only cases with recurrent attacks of repeated vomiting for at least all of one day in each attack are included.

The cyclic attack is characteristic. It has been said that to define the attack is to describe it. The child usually begins to vomit early in the morning, even before breakfast, although in some attacks the

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*Seckel[3] has also noted this as a fact in Germany.

TABLE I

SOURCE OF CASES

	TOTAL	MALES	FEMALES
Private patients	35	18	17
In consultation	9	6	3
Bellevue cases (1911-1935)	15	8	7
Staff, students (from histories)	6	2	4
	65	34	31

onset may be later in the day. The vomiting is violent, and is repeated as often as anything is taken into the stomach. Thirst soon develops, and the child cries for water, which is vomited at once or after a short interval. Even if small amounts are taken, as little as a teaspoonful at a time, vomiting occurs every hour or less, as soon as any amount of fluid accumulates in the stomach. After a day or two the vomit may be lightly blood streaked. There is a rapid loss of weight. The child becomes very weak and restless, tosses about the bed, constantly calls for water. He may sleep at night without vomiting but begins at once on waking. The eyes become sunken, the tongue dry, and the whole appearance may be most alarming. There is constipation, the urine is scanty and contains large amounts of ketone bodies, sometimes albumin and casts. In some cases extreme dehydration results. Some children complain of headache and some of abdominal pain, usually just before vomiting.

A few fatalities have been reported. Many of the children look like death. A child whom I saw only once died in an attack later. The father reported that the child had a mild attack, recovered, was given a Thanksgiving dinner, began to vomit again, and died after five days.

The duration of the attack varies; some children vomit only one day, and others may retain nothing for five days or longer (up to twelve days). The average is from two to three days. The attack ends as abruptly as it began; suddenly the child asks for food, takes it, and retains it, no matter what it may be. The loss of weight may be only two or three pounds in the short attacks, but if the vomiting persists it may amount to five pounds or even as much as ten. A new attack may supervene before the child has recovered the lost weight, so that chronic undernutrition results.

An individual child may have many attacks or only a few. Some children have one every four to six weeks for a year; others have only one a year, or perhaps only two or three during their lives. Obviously the difference in susceptibility to nervous stimuli varies in different children. I have seen several children who have had single characteristic attacks of prolonged vomiting, in whom no history of preceding attacks could be obtained, and who have as yet had no more. These are not included in this series of 65 cases, but I believe that the attacks were of the same nature as those in recurrent vomiting, and

the family history was suggestive also in at least three cases (e.g., Cases 8 and 13, Table III).* (Since submission of this paper there has been a second attack in Case 13.)

Many of the children who have attacks of cyclic vomiting become nauseated and vomit whenever they ride in trains, trolley cars, or even automobiles. The vomiting is not repeated nor of the nature of a cyclic attack, but the association of this tendency with the susceptibility to recurrent vomiting is definite. In families in which there are children or parents with a history of cyclic vomiting, some of the children become car sick or motor sick but do not have true recurrent vomiting attacks.

The age of onset in 50 patients was from six months to ten years. The average age was three and four-tenths years. In the children who have outgrown the attacks, the age at which they stopped was from four to twelve years (one case at fifteen years). Most of the patients stopped having attacks at seven or eight years of age.

The diagnosis of cyclic vomiting offers little difficulty when the attacks have been repeated. But in the first or second attack, especially when the duration is short, perhaps only one day, several other conditions may be suspected.

Appendicitis is one of these. Vomiting and abdominal pain are always disquieting, especially if there is slight fever. The normal blood count, lack of real tenderness or rigidity, and the course of the attack should always clear the diagnosis. Nevertheless, some of these patients have been operated upon, normal appendices found, and the attacks of vomiting have gone on, enhanced by the anesthesia, pain, and shock. The wary surgeon must always bear this condition in mind.

Tuberculous meningitis is not infrequently ushered in by several days of vomiting. But here the diagnosis is made all too soon, by the course of the disease.

Brain tumors occasionally cause recurrent attacks of vomiting every week or two for several months. The development of other symptoms in a short time usually clears up this diagnosis.

It must be emphasized that this type of vomiting is not the same as that associated with nervous anorexia in neurotic children. The latter vomit when forced to eat or when hurried in getting to school, but the vomiting is not repeated nor protracted.

ETIOLOGY

The cause of these attacks has long been a subject for discussion. They are precipitated by all sorts of exciting factors. Among the most frequent are the onset of an acute illness with fever, any unusual nervous excitement, an accident, an anesthetic, an exciting moving

*Ström[23] believes that certain children have only one attack in their lives, but this one is of the same nature as the recurrent attacks of other children.

picture, a trip to see Santa Claus, a shopping expedition, a fit of temper, punishment in school or at home, or a children's party, especially if any of these involve unusual fatigue. In the last case, it may sometimes be the combination of somewhat indigestible food and excitement, but I have seen attacks follow a small party at which the supper was cereal, prunes, and bread and milk served at exactly the usual time, the only abnormal factor being excitement. It is exceptional to find a story of any indigestible food, or of any especial food, or of overeating, as an exciting factor.

The child who is subject to these attacks is usually a nervous, high-strung individual, and comes from a family which is often of the same sort. There are many reasons to believe that the whole attack is a nervous explosion. It certainly does not seem to be due to any local cause in the gastrointestinal tract. The exciting factors are all those which affect the nervous system, i.e., excitement, anger, fear, shock, and fatigue. Even the sudden onset of an acute disease with fever saps the nervous reserve of a neuropathic child.

The fact that ketosis is a constant accompaniment of the attacks has led many writers to attribute them to some disturbance of metabolism. The laity almost always refer to the attacks as "acidosis," and in Europe this condition is usually described by medical writers as "acetonemic vomiting."* But ketosis appears very rapidly in any child who is not taking carbohydrate. If a single meal is missed, many children will show acetone in the urine, and it is frequently present with fever even when there is no vomiting. It must be noted, however, that cases have been reported in which acetonuria appeared just before vomiting began. On the other hand Howland and Richards,[3] Griffiths,[4] Langmead,[5] Wylie,[6] Wallis,[36] and others found that vomiting preceded the acetonuria.

Hypoglycemia is believed to be a factor by some writers. It is well known that attacks of hypoglycemia will cause serious symptoms, usually drowsiness, stupor, or even convulsions. But in these attacks there is no vomiting, as a rule, certainly not to the degree seen in cyclic cases. Convulsions are said to occur in cyclic attacks, but these must be very rare, and some doubt may be thrown on cases in which they are present, especially when they are a predominant feature rather than the vomiting (as in one of Josephs' cases[7]). I have never observed a convulsion in an attack of cyclic vomiting.

It is a matter of common experience that fatigue, loss of sleep, or nerve strain may produce an increased need for food, as shown by the increase in hunger and the ability to digest larger amounts of food than usual. It may be conjectured that nervous stimulation increases

*Ström[32] points out that Marfan, who first noted acetone breath and acetonuria, did not believe that ketone bodies were the real cause of the vomiting but a "companion of the true pathogenic toxic products" which were unknown. He has been much misunderstood in this respect.

the metabolic rate, depletes the carbohydrate reserve, and so tends to produce hypoglycemia. Fever which produces an attack in many children certainly increases the metabolic rate. Josephs[7] suggests that the patient with cyclic vomiting uses up sugar faster than the normal child. Wylie and Schlesinger[8] believe that the effects of carbohydrate withdrawal are similar to those seen in normal children. But since hypoglycemia alone does not produce continued vomiting, there must be another factor. Indeed, in cyclic attacks the blood sugar may be only moderately reduced. In several instances I have found the blood sugar to be no lower than 90 to 100 mg. per cent and that by giving intravenous glucose it may be brought up to a high normal figure without arresting the attack (Cases 16 and 29). In Josephs' experiments on a patient subject to attacks, he produced hypoglycemia with drowsiness, by starvation, but he did not produce vomiting. Seckel,[9] Hilliger, and Knopfelmacher (quoted by Josephs) were able to produce vomiting attacks by induced ketosis, but several observers, Salmonson,[10] Weichsel,[11] and others have failed to cause attacks in this way in children known to be cyclic vomiters.

Ström[33] was able to induce typical attacks in susceptible children by withdrawing carbohydrate for one or two days and then giving a highly ketogenic diet. In normal or even somewhat neuropathic children this procedure did not cause vomiting. He believes that failures by others to induce attacks have been due to the less severe or less protracted ketogenic regimen used. He suggests that children who are subject to recurrent vomiting are inordinately fond of fats and that they lose their appetites before an attack and take very little food for a day or two, which change reduces their carbohydrate intake and upsets their metabolism. He thinks that these children have a constitutional disturbance of the vegetative nervous system which affects the stomach, as well as a metabolic disturbance which reacts abnormally to carbohydrate restriction. During an attack there is a lowered assimilation of carbohydrate by which he explains the failure of glucose therapy to stop attacks in some cases. He also stresses the loss of chlorides by vomiting as adding to the metabolic disturbance, the nitrogen retention, and with the ketosis causing a vicious circle. In this monograph the metabolic aspect is stressed throughout, and little reference is made to nervous factors. The attacks which are induced by fever are regarded as a different entity. (It is difficult to believe this when one sees the same child have identical attacks brought on by fear, rage, fatigue, and also by fever.) Ström's suggestions that intake of too much fat and loss of appetite precede an attack do not correspond to my experience in the majority of cases, nor explain the sudden onset of attacks after nervous excitement with no dietary changes. Some children do have a languid day before attacks, but others seem unusually well or even stimulated.

One must grant that attacks may be produced by a severe restriction of diet in some children, but circumstances like these do not occur in a child's life. The very fact of an abrupt change in food, as in the induced experimental attacks, is not without some psychic effect and this cannot be discounted.

Wallis[36] reports a case with typical severe attacks recurring frequently, each one lasting six days. No effect was produced on the vomiting by glucose therapy. An attack was induced a few weeks later by giving only tea for breakfast and meat and vegetables at lunch. The vomiting began at once and last four days. Acetone was not present until twelve hours after vomiting began and was not always present during the rest of the attack. The blood sugar was 82 and 99 mg. per cent. This child had an increased reaction to atropine and adrenalin. Wallis concludes that the vegetative nervous system is more at fault than the carbohydrate metabolism, and states that the results obtained by sedatives point in this direction.

Weymuller and Schloss[12] found that children subject to attacks of cyclic vomiting excreted more organic acid and acetone than normal children on a high fat diet. But the children in severe ketosis did not vomit even when they were fasted and they developed a very low blood sugar (35 to 42 mg.). They also found that the ketosis diminished during starvation, perhaps because no more fat was ingested and body protein served as a source of glucose.

Schloss[13] states that, with the exception of a few experiments, attacks of typical recurrent vomiting have not been produced by the ketosis of starvation or high fat diets, even in children known to be subject to such attacks. He thinks that it seems impossible that ketosis is of constant etiologic importance in so-called recurrent vomiting. There may be a small group of cases in which such relationship exists but even this requires more convincing evidence. He points out that diabetic coma is the typical picture of severe ketosis, not violent, repeated vomiting.

It has been noted by others that the acetonuria often disappears before the attack of vomiting has ceased.

Wylie and Schlesinger[8] conclude that it does not seem that ketosis is a primary factor in periodic vomiting and that too much stress has been placed on the hypoglycemia. They say that in the presence of severe ketosis artificially produced, vomiting may occur at first but it soon ceases. Typical attacks cannot be produced in susceptible children by inducing ketosis. They found that the effect of carbohydrate withdrawal on the blood sugar was the same in normal and susceptible children. The levulose test showed disturbed liver function during the attacks but not between them.

Epileptic patients with extreme ketosis due to the ketogenic diet do not vomit, except perhaps from indigestion after an enormous amount

of fat, and this vomiting is not repeated. Ketosis in cyclic attacks may be the result of overstimulation of the nervous system plus the inevitable starvation, and too much emphasis has certainly been placed on the rôle of ketosis as a cause of the vomiting. The terms "acetonemic" or "ketotic" vomiting should be abandoned.

No demonstrable disease or lesion of the gastrointestinal tract has been present in my patients. One patient had appendicitis at fourteen, long after attacks had ceased. When there is recurrent disease of the tonsils and adenoids, their removal stops attacks of recurrent fever and illness and hence may stop cyclic attacks.

Marfan[32] reviews the various theories of the cause of the attacks. He concludes that it is possibly the result of a shock analogous to anaphylactic shock, but we are entirely ignorant of the nature of the sensitization. The causes of the attacks are so different in different

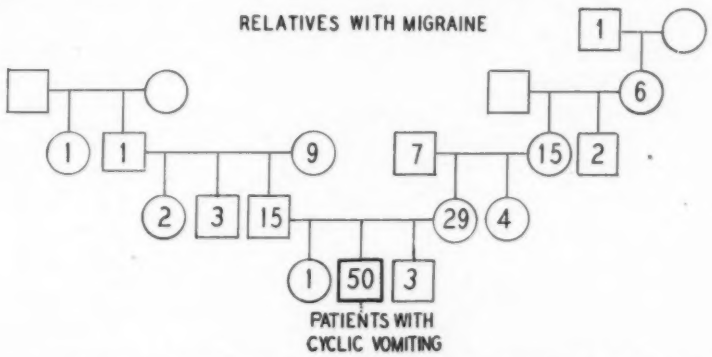

Fig. 1.—Diagram showing the cases of migraine in the families of 50 children subject to cyclic vomiting. Males are shown by squares, females by circles. Note the greater number of females affected, especially on the maternal side.

subjects and also in the same subject that their study up to the present has not found any explanation.

For many years there has been noted by many observers an association of cyclic vomiting with migraine, and in two ways. In the first place, the family history regularly shows one or more ancestors, frequently several, who have severe headaches after fatigue or on excitement. Frequently a typical migraine, with hemicrania and visual disturbances, with or without vomiting at the acme of the pain, has afflicted several relatives, especially in the maternal line. In a number of families, I have been able to observe or trace it in several generations.

In the second place, some children who have been subject to cyclic vomiting begin to have headaches at puberty, and some grow up to become victims of typical migraine. Indeed, severe headache accompanies the cyclic attack in many of the older children. This tendency has been noted by others (v.i.).

Figure 1 shows the association of migraine and cyclic vomiting in families. Table II summarizes this association in fifty cases. This must be more than accidental association. Migraine is said to occur in 5 to 7 per cent of the population. These families show a much higher incidence. Moreover, for several years I have made a careful inquiry into a large number of other family histories, on all private patients,

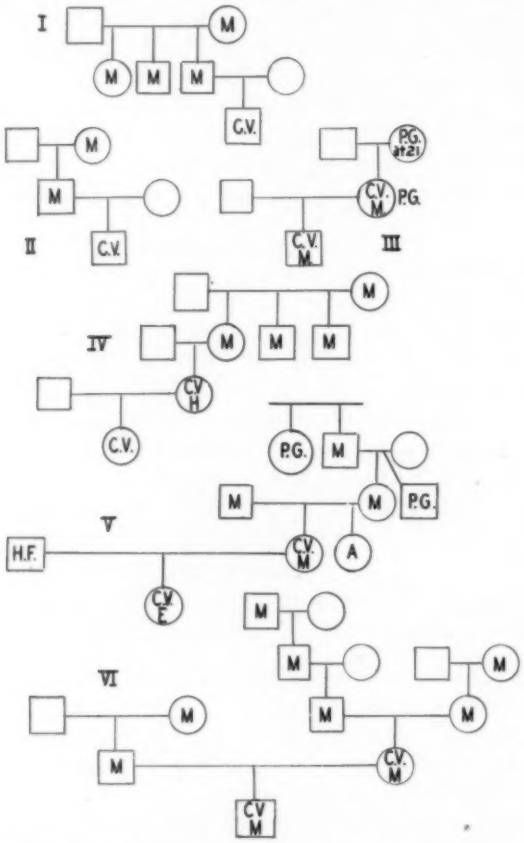

Fig. 2.—Six family trees showing types of transmission of migraine and the associated conditions. M, migraine; C.V., cyclic vomiting; P.G., premature grayness; A, asthma; E, eczema; H.F., hay fever. The last three may easily be accidental association in these families.

in regard to severe headaches. Anyone who does this will find that migraine does not often occur in a single person in a family. Either there are no sick headaches in the family, or else several members of different generations have them, provided an accurate family history can be taken. Figure 2 shows a few typical family trees.

TABLE II

RECURRENT VOMITING AND MIGRAINE IN FAMILIES

FAMILIES SHOWING ASSOCIATION	PATIENTS RECURRENT VOMITING	PATIENTS DEVELOPING MIGRAINE	RELATIVES WITH RECURRENT VOMITING	RELATIVES WITH MIGRAINE
20 in two generations	20	3	3	28
25 in three generations	25	4	4	54
4 in four generations	4	2	3	14
1 in five generations	1	1	1	8
	50	10	11*	104
No family history of migraine obtainable	5	1		
No notes on history				
Old private cases	2		*Mothers 9	
Old Bellevue cases	7		Fathers 1	
Consultation cases	1		Cousin 1	
	65	11		

In the third place, migraine attacks are precipitated by exactly the same factors as cyclic vomiting; namely, excitement, nerve strain, fatigue, and loss of sleep, which suggests a similar origin. The vomiting which comes with the most severe sick headaches is not gastric but nervous, in origin. It can be prevented by analgesics (phenacetin, pyramidon) if they are taken early enough to stop the pain before the vomiting stage is reached.

It has been pointed out that migraine sufferers usually belong to the higher intellectual and social classes. They are often prematurely gray, i.e., in the twenties or early thirties* (Kast[14]), Ulrich[15]). They are often very poor sailors, becoming seasick easily, or car sick in railroad trains or even in automobiles, like many children who have cyclic vomiting attacks.

Why should nervous fatigue cause vomiting or headache? When we know more of the exact mechanism of migraine, perhaps we can answer this question. It may well be a vasomotor phenomenon affecting the brain in both cases. Migraine has been attributed to allergic causes by the students of that branch of medicine, to epilepsy by some neurologists, to the eyes by ophthalmologists, to gastric or intestinal causes (duodenal stasis) by gastroenterologists, to endocrine disturbance (pituitary congestion or disorder of the gonads) by the endocrinologists, and to vasomotor disturbance affecting the sympathetic nervous system ("angina cranii") (Riley[16, 17]). Howland and Rich-

*At the meeting before which this paper was read, five out of six men who had marked premature whiteness of the hair admitted having severe headaches on fatigue, and two of them had had cyclic vomiting as children. Two days later I attended a luncheon at which there were again six individuals who were gray too soon, and five of them had sick headaches. Next day at a staff meeting again five out of six persons who were gray admitted having migraine. In the family histories of some patients premature whiteness of the hair is frequently reported among the parents and grandparents, but no history of headaches may be obtainable.

ards[3] attributed attacks to shock or excitement acting on an unstable nervous system, leading to a disturbed metabolism with diminished oxidation of toxic substances.

The frequent story of migraine in the families of children subject to recurrent vomiting is noted by many "older writers." Perhaps the facts obtained from good family histories were more evident to those with long experience in private practice.

Griffiths and Mitchell[18] note a tendency to migraine and other neurotic disorders in the families of children with recurrent vomiting and have noted that the vomiting attacks may be replaced by migraine in later life.

Rachford[19] also noted that sometimes cyclic patients become victims of migraine as adults.

Dunn[20] stated that these children are high-strung, easily car sick, have parents who are neurotic and subject to sick headaches and bilious attacks.

Morse[21] noted that recurrent vomiting was not infrequently replaced by migraine in later life.

Wylie and Schlesinger[8] believe that recurrent bilious attacks, cyclical, acetonemic or ketogenic vomiting, certain recurrent febrile attacks, migraine, and some attacks of recurrent abdominal pain all belong to a group of "periodic disorders," and need not be differentiated merely by a predominance of one or another symptom. In 80 children in this group, they found headache in 60, vomiting in 63, with abdominal pain in some degree in most cases, convulsions in 2, and lapses of consciousness in 7; several had slightly increased blood pressure. ("Abdominal migraine" has been described by others. In children, however, nausea and abdominal pain are not always accurately differentiated.) These authors found that the group of periodic disorders occurred in quick, alert, bright children who were slender and likely to be underweight. A large number of their parents had migraine or had had cyclic vomiting in childhood. No definite relation to allergy was found.

Bray[22] says that migraine in children takes the form of periodic attacks of vomiting with abdominal discomfort, prostration, and elevated temperature. Headache may be entirely absent and when present is less frequently unilateral than in adults.

Tallerman[2] also concludes that cyclic vomiting attacks are atypical migraine.

Wylie[6] noted the association with migraine in families and also observed children with cyclic vomiting who developed migraine in later life.

Dr. Mary O'Sullivan has kindly furnished me with the following facts from the Migraine Clinic of the Neurological Service of Bellevue

Hospital. Of 36 patients who could give good histories, 9 had had recurrent attacks of vomiting in childhood. Nineteen of the patients had children, and of these there were 4 with cyclic vomiting. Two other children were markedly car sick. One patient had a sister with cyclic vomiting. This seems more than an accidental association between these two conditions.

Balyeat and Rinkel,[23] however, found that 80 per cent of migraine sufferers were allergic and believe that both conditions are hereditary and interchangeable in the linkage. They also found many histories of cyclic vomiting attacks in childhood in migraine patients. A case is cited in which a boy who was sensitive to egg, wheat, and milk also had cyclic vomiting. But even when these foods were eliminated, he still had four attacks in a year.

Some of my patients with cyclic vomiting have had eczema or asthma, but a larger number have no evidence of sensitization nor any family history of it. The behavior of the cyclic attacks and their precipitating causes are certainly different from those of most allergic phenomena. It is true that a few patients have migraine or cyclic vomiting after ingesting definite foods (e.g., chocolate, oranges, or peanuts), but it seems to me that there is another possible explanation of this small group of cases. Sensitization is extremely common in just the class of nervous, alert, high-strung individuals who are subject to the migraine group of disorders. It may well be that a person who has both tendencies may have a migraine attack or vomiting precipitated by ingestion of a substance to which he is sensitive. In other words, his contact with the agent to which he is sensitive may merely trip the trigger which sets off the explosion.

Tallerman[2] found a definite family history of allergy in 45 per cent of 40 children subject to cyclic vomiting but in only 25 per cent of 40 control children. Apart from this, no definite evidence of allergy was found, and skin tests failed to reveal sensitivity to common foodstuffs.

Neff[27] found visceral sagging and fatigue in children subject to cyclic vomiting and noted that they are rare in hospitals, but are usually seen among the well-to-do classes of neuropathic tendency.

Talbot and Brown[28] found faulty body mechanics as a background in most of their patients with cyclic vomiting. They obtained good results with braces to correct posture and with rest periods of one-half to one hour after each meal. I wonder whether the rests which helped to avoid fatigue were not as important as the braces. Since some patients are undernourished as a result of repeated losses of weight, it is inevitable that many of them will have bad posture, which is so common in underweight children. But not all of these children are underweight. In the 26 private patients shown in Table III only a

few had poor posture, and the nutrition of only four was definitely poor. Six of them were plump or fat.

It must be pointed out that histories in this type of disorder are not always easily obtainable, for many reasons. In the hospital class few patients know much about their parents, many of whom are in Europe or are not alive. Cyclic vomiting is easily forgotten after it has ceased and is often put down to acute indigestion by the parent. Migraine also has a tendency to cease at the ages of 40 to 50 years and may be forgotten by other members of the family, or its real nature not understood. Many attacks of migraine are attributed to biliousness, to indigestion or constipation, to the eyes, or to sinusitis. In private practice much more reliable histories can be elicited, but even in this class it may be necessary to interview some older members of the family, a grandmother or a great-aunt, to find out that sick headaches have occurred in several generations and that the mother of the patient with cyclic vomiting also had similar attacks as a child. How many of us know whether our great-grandmothers had migraine or even whether our mothers vomited repeatedly between the ages of two and six years?

The uncertainty of the nature and the cause of migraine is evident. It is believed, however, that it is definitely a hereditary condition.

Snyder[24] says, "At the other extreme would be a character in which heredity is the variable influence, no environment of sufficient potency to alter the expression of the heredity being readily available at present. Such a character is migraine (a type of sick headache) where a person develops the condition regardless of environmental influences if he inherits the factor for it, and where migraine does not develop if the factor is not inherited, the factor for this condition being a simple autosomal dominant.

"Migraine, or unilateral sick headache, is sometimes included in the allergies, but is usually transmitted specifically, and is probably due to a separate dominant gene."

Buchanan[25] studied 127 families in the Mayo Clinic and found that migraine is definitely transmitted in a ratio approximately three to one as in all dominant mendelian characters.

Stieglitz[26] says that migraine has long been known to be familial and much has been written confirming this fact.

Kast,[14] who has studied migraine for many years, believes that it is definitely hereditary, mostly through the mother, and that cyclic vomiting is a childhood equivalent.

Migraine is a definite entity. An individual either has it or does not have it, and those who do not may have no conception of what it means to have that type of headache. Its victims know all too well what to expect after fatigue or excitement.

TREATMENT

From the above it is evident that many independent observers have noted the association of migraine with cyclic vomiting, in families and in individuals. A further fact points in the same direction; i.e., the result of similar treatment.

The usual treatment of cyclic vomiting is to give small amounts of water, soda water, or ice in the effort to allay the thirst and to arrest serious dehydration. This effort is wasted, for fluids are vomited until the attack is over. Saline or glucose is commonly given subcutaneously, intravenously, or rectally in severe cases. Saline solution by rectum is of limited use in childhood. Carbohydrate is also given in the form of lollipops, crackers, or cereal by mouth, or glucose by infusion or by rectum. Only a small amount of glucose can be absorbed in the last way even in cooperative children. Of course, it can be given intravenously to any desired amount.

Under this treatment, the attack lasts from two to four days, and the results are not striking. The children remain desperately prostrated, restless, and lose much weight, which may not be regained before the next attack (Cases 16, 28, and 29).

But if the nervous system is treated as the real seat of the explosion, the results are very different. A large dose of a sedative, such as bromide, amytal, luminal by rectum, or codeine subcutaneously, will often abort the attack so that it lasts only a few hours, or at most a day, instead of from two to five days.

For about fifteen years I have used the following procedure: As soon as the child vomits, he is given an enema of strong soapsuds. When the mother or nurse is certain that this is evacuated, that is, after the child has been urged to expel all of it two or three times (usually about one hour after the cleansing enema), a solution of sodium bromide is given by rectum. This can be done most easily by using a small infant-sized rectal syringe. The total volume of the solution should not be over two ounces. About ½ grain per pound is an effectual dose, 20 grains for a 40 pound child, 40 grains for one weighing 80 pounds.

After the injection is given, the buttocks are pressed together, with the child lying on his side, and he is urged to retain the medicine. Nothing is given by mouth, until two or three hours after the bromide, then dry toast or crackers are offered. If these are vomited, a second dose of bromide is given, and solid food is offered an hour or two later. No liquid is given by mouth until some solid is retained, then orange juice sweetened with sugar or lactose in very small amounts may be started. By this time it is afternoon, and, if liquid is retained, a light supper of thick cereal with very little milk is given. A mild laxative is given at 10:00 or 11:00 P.M. for a few nights. The next day the

TABLE III

NAME AND SEX	AGE			ATTACKS OF VOMITING					OTHER SYMPTOMS	NUTRITION	RELATIVES			
	PRESENT (YR.)	ATTACKS BEGAN (YR.)	ATTACKS STOPPED (YR.)	INTERVAL	NUMBER	CAUSE	DURATION				CYCLIC VOMITING	MIGRAINE	PREMATURE GRAY HAIR	CAR SICK
							WITHOUT SEDATIVES	WITH BROMIDE						
1. C. L. F	7	1½	–	Irregular	2-3	Excitement Automobiling	Bromide all attacks	6-8 hr.	Placid Eczema Hemorrhage of newborn Car sick	Good	M	M* GM (maternal) GF (maternal) GGM (maternal)	A (paternal) A (paternal) U (maternal)	
2. N. J. F	7½	2	–	Several mo.	2-3	Fever	Bromide all attacks	6-12 hr.	Placid Eczema	Good		A (paternal) GM (paternal)	A	
3. D. C. F	8	6	–	6 mo.	3	Fear Excitement	Bromide all attacks	12-36 hr.	F. deserted family Nervous	Fair Slender	3 cousins vomit after any excitement	A (maternal)	A	
4. M. S. F	9	4	–	Irregular	Many	Parties Excitement	2 days	2-6 hr.	Motor sick Spoiled	Thin	Cousin (?)	GM (maternal)		
5. G. G. F	12	2	9	12 mo.	Several	Fever Excitement	3-4 days	3-4 hr.	Eczema	Plump		F		
6. S. H. F	13	2	12	At 2, 3, 10 and 12 yr.	4	Party Movies	1 day	Few hours Bromide; codeine necessary for 1 attack	Car sick Highstrung	Good		M	M GM (maternal)	
7. E. M. F	13	2	5	4-6 mo.	Several	Excitement Heat	1-2 days	½ day	Spoiled	Good		M GF (maternal) Br	GF	

TABLE III—CONT'D

8. E. W. F	14	7	7		Only one definite attack	Excitement and fatigue		2 days Bromide; codeine needed	Headache unilateral at 9½ Placid	Fat		GM (maternal)	GF (maternal)	M very seasick
9. F. G. F	16	2	12	Irregular	Many	Fever	2-3 days	½ day	Headache on fatigue after puberty Hypothyroid	Fat		F GM (paternal)		
10. E. S. F	18	2	13	Irregular	Many	Fever Excitement	4 days	Few hours	Car sick Headaches at 15 not typical	Good	M	GM (maternal) 2 GU (maternal) GGM	GM GGM	M
11. V. A. F	18	4½	13	1-6 mo.	Many	Excitement	4-5 days	Few hours	Headaches after puberty Very tall	Thin	M	M GGM (maternal)	M	
12. E. McK. F	20	2½	8	Irregular	Many	Fever Fatigue	2 days	Few hours		Good		M GM (maternal)		
13. D. W M	5½	4½	-		1	Auto ride and fever	1 day	Not used	Frequent headaches	Fair		M A (maternal) GM (maternal)		
14. W. K M	6	2	-	Several mo.	3-4	Infections Fear	Bromide always	6-8 hr.		Good	F	F		
15. R. C. M	7	3	-	4-18 mo.	5-6	Fever A party Movies	3 days	Bromide stops attack promptly	Car sick Spoiled	Excellent		F GM (maternal)	F at 19	

TABLE III—CONT'D

NAME AND SEX	AGE			ATTACKS OF VOMITING					OTHER SYMPTOMS	NUTRITION	RELATIVES			
	PRESENT (YR.)	ATTACKS BEGAN (YR.)	ATTACKS STOPPED (YR.)	INTERVAL	NUMBER	CAUSE	DURATION				CYCLIC VOMITING	MIGRAINE	PREMATURE GRAY HAIR	CAR SICK
							WITHOUT SEDATIVES	WITH BROMIDE						
16. A. P. M	7	2	–	1-4 mo.	Many 15-20	Fatigue Excitement Anger Fever	3 days + "with infusions of glucose"	5-8 hours usually Codeine, in some attacks	Spoiled	Thin		M F GM (maternal)		
17. H. B. M	8½	3½	–		2-3	Fever "nerves"	1-2 days	None given	Wilful Frequent headaches Car sick	Thin	M	M GM (maternal)	GM	
18. A. C. M	10	4	6½	Several mo.	4-5	Infections	1-3 days	Few hours	Spoiled Self-satisfied	Fat		M GM (maternal)	M	
19. M.W. M	10	3½	8½	3-6 mo.	Several each year	Fever Fatigue	2-3 days	1 day Codeine in some attacks		Good	M	M GM		M
20. G. H. M	10	3	?	4-6 mo.	Several	Excitement	2 days	Under 1 day		Slender	Br M GM (maternal)			Br
21. H. E. M	11	3	6	1 yr. +	3-4	Infections	Bromide always	8-12 hr.	Nervous anorexia	Good	M	GM (maternal)		M
22. J. M. C. M	15	3	? (Moved away)	2-3 mo.	Many	Infections	2-3 days	12-18 hr.	Spoiled	Good			M gray Very neurotic No intelligent history	

TABLE III—CONT'D

23. H. B. M	15	3	10	1-6 mo.	Many	Fever Fatigue Excitement?	1-2 days	6-8 hr.	Headache with vomiting attack	Good		F GM (paternal)	F	
24. M. C. M	15	5	10	6-12 weeks	Many 8 in 1 yr.	Fatigue School	1-2 days	"None tried" (Seen first at 11 yr.)	Headaches at 10-11 Car sick	Fair	M	M GF (maternal)	M at 30 GF at 21	M
25. F. B. M	15	4	8	4-6 mo.	2-3 in 1 yr.	Tonsillitis Excitement Chilling	2 days	Not seen in attack	Family poor	Poor -10%		F U (paternal) A		
26. H. O. M	18	3	12	1 mo. 1 yr.	Many	Excitement Fatigue	3 days	½-1 day	Migraine after 13 Wilful	Excellent	M	GM M GM (maternal) GGM (maternal) GF (maternal) GGF (maternal) GGGF (maternal) F GM (paternal)		M

*M, mother; F, father; GM, grandmother; GF, grandfather; GGM, great grandmother; GGF, great grandfather; U, uncle; A, aunt; Br, brother.

regular food is given in half portions and by the third day a full diet. The child stays in bed three days, then is gradually allowed up, and is often as good as new, with little or no weight loss.

Occasionally there is one vomiting the second morning. This may not need treatment, but if repeated, the enema, bromide, and solid food should be given again.

In cases which do not yield to bromide after two doses, or those which are likely to be severe, judging by past attacks, codeine sulphate in full doses (1/100 grain per pound) is given hypodermatically. A child of 50 pounds receives ½ grain, one of 75 pounds, ¾ grain. These are larger doses than are usually given but are quite safe.

This plan is simple and effective. I have seldom seen an attack which failed to yield to it in less time than the usual attacks treated without sedatives (Table III).

It is not necessary to give parenteral fluids to any patient who is seen early. Hypodermoclyses and infusions are somewhat painful and upsetting to the nervous child, to whom they seem heroic procedures. They may easily add to the nervous excitement and prolong the attack. In my opinion they have no place in the treatment of this condition, except in cases which have gone on for several days to dangerous dehydration. I have never had to resort to infusions or hypodermoclysis in private patients and rarely in the hospital patients. Removal to a hospital also tends to produce nervous excitement. Since the attacks are likely to occur again and again, hospital treatment becomes an economic burden.

This principle of treatment by sedatives is mentioned by some "older writers" but is conspicuous by its absence from the newer textbooks and papers. Holt's textbook[29] (tenth and previous editions) says that when the vomiting has begun, nothing seems to have the slightest influence in controlling it. Parenteral fluids and glucose are advised merely to support the patient through the attack, or even bicarbonate intravenously if acidosis cannot be controlled.*

Seckel[9] states that prompt recovery occurs after sugar therapy, but, in the cases reported, one had vomited five days, and the other, two days before treatment. Perhaps they were ready to stop.

Ström[33] and Wallis[36] were unable to arrest attacks with glucose. Griffiths and Mitchell[18] advise glucose by rectum or intraperitoneally with insulin, and blood transfusions. They suggest cocaine hydrochloride by mouth and morphine in divided doses if nothing else stops the vomiting. They have found bromide and chloral by rectum unavailing.

Chapin and Royster[30] recommend bicarbonate and glucose by rectum and water or soda water freely by mouth in the hope that some of it may be retained.

*The last edition (1936) mentions the use of bromides.

Wylie and Schlesinger,[8] who are quoted above as believing that cyclic vomiting is not due to ketosis, nevertheless recommend treatment with parenteral fluids and glucose for the attacks.

Ross and Josephs[31] treated a negro child who had attacks of drowsiness, convulsions, and vomiting and who showed hypoglycemia and ketosis, by infusions of glucose, and were able to stop the attacks promptly. The description of the attacks is not that of typical cyclic vomiting, in which drowsiness and convulsions are not characteristic.

Parmelee[37] in Brennemann's *Practice of Pediatrics* says that nothing seems to have any influence on the vomiting and that it is best to omit all attempts to stop it by the use of drugs. The usual treatment by parenteral fluids and glucose is advised. . . . I cannot understand the point of view which suggests that it is undesirable to attempt to control the vomiting. These attacks are always exhausting, the loss of weight is great, the child is in a pitiable condition, which often becomes alarming, and he may even die. He who has not used full doses of sedatives can have no appreciation of their almost magical results.

In the last generation several authors mention the use of sedatives.

Griffiths[4] advised morphine in a severe case. It was not used, and the child died. Koplik[38] says that codeia is the only remedy that had any effect in controlling the attack in his hands. Rotch[33] states that bromide and chloral may be given by rectum, if the attack lasts over twenty-four hours, and that even morphine may be necessary. Morse[21] says that the old treatment by bromide and chloral by rectum, with morphine for the most severe cases, is more likely to be helpful than the treatment of acid intoxication. Dunn[20] advocates bromide and chloral by rectum in the severe or prolonged cases, and even morphine as a last resort. Tallerman[2] suggests luminal for children who have severe headaches during the attacks, but does not mention its effect on vomiting.

When we come to the interval treatment of the child, again various plans are suggested, and these are often contradictory.

Those who believe that the underlying fault is the inability to digest or metabolize fat suggest a diet low in fats and high in carbohydrates between the attacks. Among these are Tallerman, Holt and MacIntosh, and Morse. On the other hand, a diet low in carbohydrates has been recommended.

If the underlying cause is a hereditary, inescapable tendency, there seems little use in trying to avert the attacks by restricting one food element, especially since it remains to be proved that inability to metabolize fat lies at the root of the disorder. Most writers are agreed that the child must be spared fatigue and must have more rest than the average child. Excitement, fear, and anger should be avoided as much as possible, but who can be entirely spared these things? A full, balanced, nourishing diet which builds up the child's muscles and fat

seems to me the most logical one, so that he may have some reserve strength to meet emergencies and reserve weight in preparation for possible loss in future attacks. If fats are eliminated from the diet too much carbohydrate or protein must be given. Large amounts of the former predispose to infections. Too much protein is somewhat hard on the digestion. There is no definite evidence that restriction of fats has any effect on diminishing the number of attacks. These children do very well on a normal diet and have fewer attacks if a careful regime is planned, one which avoids fatigue and excitement as much as possible. It is important also to establish a regular morning bowel habit, and to be sure that all of the child's hygiene is free from gross faults.

Table III shows some of the facts on a group of private patients who have been observed in attacks of cyclic vomiting, with a few whose history showed characteristic attacks in the past. The age incidence, exciting causes, and frequency are indicated. The duration with the usual treatment or with glucose is seen to be longer in every case than when bromide by rectum was used. Many of these children were markedly car sick, most of them high-strung, several badly spoiled. Their nutrition was not notably bad, with a few exceptions, and in these cases the effect of spoiling, and economic stress (Cases 3 and 25) were perhaps as important as the loss of weight during attacks, although in a few instances (Cases 4 and 16) in which the attacks were severe and frequent, this was a definite factor. The posture and the general physique of nearly all of these children were good, some of them being splendid specimens. One had hypothyroidism. Several of them have passed puberty, and a few have developed more or less characteristic headaches. The incidence among relatives of cyclic vomiting, migraine, car sickness, premature graying is shown. In some families the grandparents were dead, and no facts could be elicited about any relative except the parents. It is significant, however, that in every family of this group one or more relatives had cyclic vomiting or migraine, and several had the associated symptoms. If one takes histories of the same number of families at random no such incidence will be found.

These cases were selected from the total group studied merely because I had personal contact and have fairly complete histories which could not be obtained from the older private records, from consultations, or from the hospital records. Although some children had few attacks, each one was characteristic. Two children with but one attack each are included in this table, but not in the total of sixty-five mentioned.

CASE REPORTS

CASE 8.—E. W. had a typical, severe attack at seven years after excitement and fatigue. Bromide alone did not control the vomiting but it ceased promptly after a full dose of codeine on the second day. She developed unilateral headaches at the

age of nine and one-half. Her family history is suggestive, so that it seems likely that this child had the tendency to vomit but that the exciting cause had to be unusually strong. She lived in the country, led a model, sheltered life, and has always been large and somewhat overnourished, though not very fat.

CASE 13.—D. W. is only five and one-half years old and has had what was perhaps the first attack. He may have more. (He had his second attack after this paper was submitted.)

The histories of the following cases illustrate some of the facts mentioned, in more detail than is shown in the table.

CASE 16.—A. P., aged seven years. His father and father's mother had severe headaches; they were not gray. His mother had nervous headaches after fatigue. The entire family was high-strung; the patient was wilful and spoiled.

Attacks of vomiting began at two years, usually following excitement or fear. Lately he has had attacks when his mother has left him for the day, when a fire engine stopped outside the house, after fatigue of playing too hard, or with fever.

Vomiting in the early attacks was severe, frequent, and persistent for three days. In the first two attacks the patient was taken to the hospital on the first day and given intravenous glucose, but vomited for two days more in each attack.

He was seen in consultation at the onset of the third attack (at two years eight months). Two physicians advised glucose infusions. The hospital room was engaged and a car ready to take him, but bromide was given by rectum, and he ceased vomiting.

He was seen next at four years and four months. He had had several attacks, all controlled by bromide in from five to ten hours. After a tonsillectomy at three years he began vomiting, but bromide controlled it promptly. A severe attack at five and one-half years came on after fatigue and excitement. He would not retain the bromide given rectally, and when seen the second day he was in very poor condition, dehydrated and extremely restless. It required three doses of codeine of ⅜, ½, ½ grains in seven hours to stop the vomiting.

At five years ten months a severe attack came on after excitement over his father who had had a heart attack. He would not retain bromide but did retain an amytal suppository and vomited only a few times in the next six hours, after which he stopped.

CASE 27.—H. A., a male, seven years old. (Not shown in Table III.)

Family History.—The entire family is high-strung. One sister is very nervous but has never vomited. The mother had cyclic vomiting as a child and now has migraine attacks, and she is prematurely gray. Her sister has migraine. The mother's mother is not living, but had been prematurely gray and the mother's father had snow-white hair as a young man; no history of headaches was obtainable.

Past History.—The patient had been a healthy boy except for occasional respiratory infections. At two years he began to have vomiting attacks, and between three and four years these were frequent. They came on with the onset of any febrile illness or after fatigue or excitement. They never lasted over one day but were promptly stopped by bromide by rectum.

He had no attacks after four years until three weeks ago (December, 1934), when after the excitement of Christmas, he had an attack in which he vomited for three days. His mother had forgotten his earlier attacks and thought it was indigestion and did not report it.

Present Attack.—The child was taken downtown shopping all day, and visited Empire State Building tower. He was tired and listless at night. The next day

he had a slight cold and a temperature of 101° F. Vomiting began at noon and was repeated all evening and night, every hour or two. The next morning he was still vomiting and was prostrated; his temperature was 101° F., and he had coryza and a red pharynx.

Bromide, 30 gr., was given by rectum at 10:00 A.M. He vomited only once more and took solid food in the afternoon. He was well the next day.

This case illustrates the prompt effect of bromide which stopped every attack in which it was given. In the single attack in which it was forgotten, the boy vomited three days.

CASE 28.—M. W., twelve years of age, of English descent, was seen in the ward at Bellevue in February and April, 1935. (Not in Table III.)

Family History.—One sister has cyclic vomiting. One brother does not. The mother had cyclic vomiting as a child, and now has migraine attacks. The mother's sister, mother, and grandmother all had sick headaches.

Past History.—Irrelevant except for attacks of vomiting which began at eight months, recurring every two to three months, always lasting two or three days. They occurred after excitement or fever.

On Feb. 11, 1935, the child caught cold, had a slight fever. Vomiting began at night. She vomited all day February 12 and everything was ingested. She had a severe headache. Vomiting continued all morning February 13, and she was admitted to the hospital at noon. She was drowsy, restless, and retched frequently. The blood sugar was 101. The blood carbon dioxide was 50 volumes per cent, temperature 101° F., acetone in the urine, 3 plus.

She was given clyses of saline and two infusions of glucose by the house staff, but was also given sodium bromide 30 gr. rectally. The vomiting stopped after the bromide and the child retained solid food.

On February 14 the temperature was 101° F. The patient was somewhat hysterical, but there was no vomiting. On February 15 the temperature was normal and the patient was well and hungry. When the patient was discharged, the mother was warned about avoiding the exciting causes and was told to bring the child back at the very onset of the next attack.

On April 7 the child rose early and went to mass without breakfast. She ate nothing until noon when she was very hungry and ate a large dinner. She had supper at the house of a friend and went to a late moving picture.

On April 8 the child vomited on waking and continued all day, retaining nothing. She objected to going to the hospital so was not brought in until the next day. She vomited all morning, April 9, and was admitted to the hospital at noon. Her temperature was 99° F. There was moderate prostration and restlessness. Blood sugar was 94. The blood carbon dioxide was 56 volumes per cent. There was strong acetonuria.

The house staff had been warned not to give any parenteral fluids. Sodium bromide, gr. 45, was given by rectum, and she was fed toast by mouth. There was no more vomiting, and the patient was discharged in two days.

Note that the second attack yielded just as promptly as the first although no glucose was given.

The following case illustrates the futility of the usual treatment which is directed at the ketosis alone.

CASE 29.—T. R., male, aged five years, was admitted to the Bellevue Hospital in November, 1924. No family history was obtained. Six months ago he had an attack in which he vomited all food for ten days. Two months ago he had a similar attack lasting several days.

Present Attack.—Three days before admission he began vomiting and had epigastric pain. Vomiting was repeated on the least motion of the child. He was thin, pale, listless, with sighing respirations. There was doubtful epigastric tenderness. His temperature was normal. The urine contained a large amount of acetone.

He continued to vomit after admission. He was given saline hypodermoclyses and four intravenous infusions of glucose between the fourth and eighth days of the attack and a transfusion on the eighth day. On the fourth day the blood sugar was 101; the blood carbon dioxide was 37 volumes per cent. On the eighth day the blood sugar was 158; the blood carbon dioxide was 48 volumes per cent. He continued to vomit even with a high blood sugar until the twelfth day, when he stopped and made an uneventful recovery.

CASE 30.—E. C. The great-grandmother and great-aunt (maternal) had sick headaches. The mother's father was prematurely gray and neurotic.

The child never had any characteristic vomiting attacks but is so car sick that it is impossible to take her in a motor car. After a short ride she always vomits. General nutrition and mode of life are excellent. The family lives very quietly in the country. This is the kind of child who might have had cyclic attacks if her general hygiene had not been so good, and if she had not been carefully protected against the usual exciting causes.

SUMMARY

Recurrent or cyclic vomiting is a definite clinical entity seen in certain children, more often in private than in hospital practice in this country. The children usually come from families whose members are high-strung, of above average intelligence, and in which there is a definite family tendency to migraine, cyclic vomiting, and prematurely gray hair, especially on the maternal side. A number of children with cyclic vomiting grow up to have migraine in adolescence or young adult life. Some become gray before they are thirty. The effect of treatment with sedatives or analgesics in both conditions also points to a conclusion that recurrent vomiting is a childhood equivalent for migraine.

Ketosis accompanies the attacks, but it remains to be proved that it causes the vomiting. Treatment of the ketosis with rectal or intravenous glucose has no effect on the vomiting, and ketosis may be disregarded except in neglected patients who have been vomiting several days. Administration of bromide by rectum, or codeine hypodermatically will abort most attacks in a few hours.

In the intervals between attacks the child should be given a full nourishing diet. There is no reason for restriction of fats. He should be spared fatigue and excitement as much as is humanly possible. He needs extra rest periods and a carefully planned daily regime.

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THE INTERPRETATION OF X-RAY FILMS IN THE DIAGNOSIS OF RICKETS

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BETWEEN 1933 and 1935, in the course of some growth studies carried on jointly by the Research Laboratory of the Children's Fund of Michigan and the Children's Hospital, x-ray films of a series of infants were made at various stages of their development. Though the intent was not to make a study of rickets, its presence or absence was, of course, an important point in the reading of the films. This reading was done by the three clinicians and two roentgenologists of the Children's Hospital staff who collaborated in the study, each making his own reading, which was recorded separately. Later, because of interest aroused by differences in these readings, a number of the films were submitted to Dr. Martha Eliot, of the Children's Bureau, and to Dr. Philip C. Jeans and Dr. Carl L. Gillies, of Iowa State University, who were good enough to give their interpretations of them. Analysis of these various ratings has seemed to us of sufficient interest to justify their publication, with some comments.

Personnel.—The clinicians, Thomas B. Cooley, B. M. Hamil, and Marsh W. Poole, and roentgenologists, Lawrence Reynolds and E. R. Witwer, of the Children's Hospital staff, have all had a fairly wide experience in the study of films for rickets. The studies in this field of Eliot, and of the Iowa clinic, represented here by Jeans, clinician, and Gillies, roentgenologist, reporting jointly, are well known.

The Films.—Each of the participants had read a different number of the films made in the course of the study. The present comparison (Table I) is based on the readings of a group of 116 films rated by

TABLE I

SEVEN PHYSICIANS' RATINGS FOR RICKETS OF 116 X-RAY FILMS

RATING	TYPE OF RICKETS	PHYSICIANS						
		I	II	III	IV	V	VI	VII
A	Normal	52	50	56	55	66	48	52
B	Slight	29	53	48	47	23	32	47
C	Moderate	7	10	9	11	23	6	12
D	Active		1	3	1	3		4
E	Healed							1
	Not read	28	2		2	1	30	

Jeans and Gillies, all of which had been read by the majority of the Detroit group, and all but one by Eliot. These films were made at dif-

From the Research Laboratory of the Children's Fund of Michigan and Children's Hospital of Michigan.

TABLE II

INTERPRETATION OF X-RAY FILMS BY DIFFERENT READERS*

CASE NO.	PHYSICIANS' RATINGS						
	I	II	III	IV	V	VI	VII
1	A	A	A	A	A	A	A
	B	C	B	B	B	B	B
2	A	B	A	A	A	A	A
	B	B	B	C	B	B	A
	B	B	B	B	B		A
3	A	A	A	A	A	A	A
	B	B	B	C	B	B	B
		A	A	A	A		
4	A	A	A	A	A	A	A
	B	B	C	B	B	B	A
		B	B	B	C		B
5	A	A	A	A	A	A	A
	A	A	A	A	A	A	A
		B	B	B	C		B
6	A	A	A	A	A	A	A
	B	C	C	B	B	B	B
	C	C	C	C	B	C	B
7	A	A	A	A	A	A	A
	A	B	B	A		B	A
	A	B	B	A	A	B	B
8	A	A	A	A	A	A	A
	B	B	A	A	A	A	A
	C	C	D	C	C	C	B
			D		C		D
9	C	C	D	C	C	C	D
		B	B	B	C		D
10	A	A	A	A	A	A	A
	A	B	B	A	D	A	B
		B	B	B	B		B
11	A	A	A	A	A	A	A
	C	B	B	C	B	B	B
		B	B	B	C		B
12	A	A	A	A	A	A	A
	B	B	B	B	A	B	B
13	A	A	A	A	A	A	A
	B	B	B	B	A	B	B
14	A	A	A	A	A	B	C
		A	A	A	C		E
15	A	B	A	A	A	B	B
	B	B	B	B	C	B	C
		B	B	B	C		C
16	A	A	A	A	A	A	A
	A	A	B	A	A	B	B
17	A	A	A	A	A	A	A
	A	B	A	B	A	A	B
		B	B	B	A		B
18	A	A	A	A	A	A	A
	B	B	B	B	A	B	B
19	A	A	A	A	A	A	A
	B	B	B	B	C		B
		C	C	C	C		B
20	A	A	A	A	A	A	A
	A	A	A	B	A	A	A
		A	A	A	A		A
21	B	A	A	A	B	B	A
	B	B	B	B	B	B	B
22	B	B	B	B	D	B	D
		C	C	C	C		C
23	A	A	A	A	A	A	A
	A	A	A	A	A	A	A
		B	B	B	B		B
24	A	A	A	A	A	A	A
	A	A	A	A	A	A	A
25	B	B	A	A	A	B	A
	C	C	B	B	B	C	C
26	A	A	A	A	A	A	A
	B	B	B	B	B	B	B
27	A	A	A	A	A	A	A
	C	C	C	C	B	B	B
28	A	A	A	A	A	A	A
	B	B	B	B	A	B	B
29	A	A	A	A	A	A	A
	A	B	A	C	A	A	A
		A	A	A	A		B
30	A	A	A	A	A	A	A
	B	B	C	B	B	B	B
	B	B	B	B	B	B	B
		B	B	B	B		C
31	A	A	A	A	A	A	A
	A	B	B	B	C	B	B
			B		C		C
32	A	A	A	A	A	A	A
	A	B	A	B	A	A	A
		B	B	B	B		C
33	A	A	A	A	A	A	A
	B	C	B	C	C	C	C
	C	D	C	D	D	C	C
		B	B	B	C		C
34	B	A	A	B	A	B	A
	B	B	B	B	C	B	B
		B	B	B	C		
35	B	B	B	B	C	B	B
		B	B	B	B		B
36	A	A	A	A	A	A	A
	B	B	C	B	C	B	B
		B	B	B	C		B
37	A	A	A	A	A	A	A
	A	A	A	A	A	A	A
		B	B	B	A		B
38	A	A	A	A	A	A	A
	A	A	A	A	A	A	A
		B	B	B	A		B
39	A	A	A	A	A	A	A
	B	B	B	A	B	B	B
		B	B	B	B		B
40	A	A	A	A	A	A	A
	B	A	B	B	A	B	B
		B	B	B	C		C
41	A	A	A	A	A	A	A
	B	B	A	B	A	A	B
		B	B	B	A		B
42	A	A	A	A	A	A	A
	A	B	B	B	A	A	B
		A	A	A	A		B
43	A	A	A	A	A	A	A
	B	B	B	B	B	B	B

*A—Normal
B—Slight rickets
C—Moderate rickets
D—Active rickets
E—Healed rickets

ferent age periods from forty-three infants, all of whom were thought at some period by one or more of the Detroit group to show x-ray evidence of rickets. The films generally were agreed to be excellent. Table II gives all the readings of these films, and shows the considerable variations between the interpretations of different readers.

Study of these tables and observations of the attitudes of the several readers indicate that there would be practically no disagreement with regard to rickets advanced enough to be of clinical importance, and that differences in grading between "mild" and "moderate" rickets were chiefly matters of individual terminology, and not of especial significance. Readings of "active" and "healing" rickets might have been interesting if there had been enough of them for comparison.

The really important feature in the various readings was the conflict of opinion as to the presence or absence of evidence of rickets in individual films. Of 79 films read by one or more of the group as showing rickets there was a difference of opinion in regard to 32. Table III is

TABLE III

THE NUMBER OF FILMS IN WHICH EACH PHYSICIAN'S READINGS DIFFERED FROM THAT OF THE MAJORITY

	I		II		III		IV		V		VI		VII	
	R	N	R	N	R	N	R	N	R	N	R	N	R	N
Film 2:1			6											
2:2														6
2:3														5
4:2														6
7:3		4						4		4				
8:2	5		5											
10:2		4						4				4		
12:2										6				
13:2										6				
14:2									3				3	
15:1			4								4		4	
16:2					4						4		4	
17:2			4				4						4	
17:3										4				
18:2										6				
20:2							6							
21:1	4								4		4			
25:1	4		4								4			
28:2										6				
29:2			5				5							
29:3													4	
31:2		6												
32:2			5				5							
34:1	4						4				4			
37:3										4				
38:3										4				
39:2								6						
40:2				5						5				
41:2						4				4		4		
41:3										4				
42:2		4								4		4		
42:3													4	
	17	18	33	5	4	4	24	14	7	57	20	12	23	17
Films read	25		32		32		32		31		24		32	

designed to bring out these differences by showing the number of films in which each one's reading differed from that of the majority, using a point scoring system showing with how many others each was in disagreement. Thus, a score of 6 shows that the individual reader disagreed with all the others; 5, that he was one of two, etc. In a few instances an isolated reading was scored only 4, because the film was not read by all. The totals show the number of readings of the rest of the group with which the individual differed, R indicating a reading of rickets, and N, normal. Thus V, in a total of 31 readings, disagreed with 64 of 146 readings of the others, 57 of the 64 being in favor of rickets as against V's reading of normal.

Comment.—There is no assumption that agreement with the majority is any evidence of the correctness of the individual reading. The striking thing to be derived from a study of Table III is the remarkable lack of agreement between any two in their minority readings. Observer V was alone in 8 of 14 readings, all of these being "normal"; VII was alone in 4 of 9; V and VII, who had the highest number of minority readings, agreed only once; II, IV, and VII all tended to read in favor of rickets, but in only 7 of their 18 readings was there any agreement. Such analysis of the table could be extended. It seems to indicate that these seven observers, each presumably as experienced and competent as the average person who undertakes to determine the presence or absence of rickets from x-ray films, cannot have been governed by any common, generally recognized criteria for the determination of minor

TABLE IV

THE PERCENTAGE OF MINORITY READINGS FOR EACH OBSERVER AND THE PERCENTAGE FOR AND AGAINST RICKETS

	I	II	III	IV	V	VI	VII
Rickets	16	21.8	3.1	15.6	6.1	20.8	18.7
Normal	16	3.2	3.1	9.4	39.0	12.5	9.4
Total	32	25.0	6.2	25.0	45.1	33.3	28.1

degrees of this disease process. Such a situation is not, of course, unique. Doubtless very similar disagreement would be found in the reading of films for any one of a number of pathologic states. The use of the x-ray for diagnosis has by no means attained the status of an exact science. Its value in any particular study must depend on the interpreter's understanding of the physical characteristics and limitations of the method itself, and of the pathologic states in question and the physical effects to be expected from them. To attain the highest degree of accuracy in the reading of films requires a combination of the knowledge of the roentgenologist, the clinician, and the pathologist, and either roentgenologist or clinician will be successful in proportion to his competence in all these fields. It is to be assumed that each of

these seven observers has made painstaking effort to qualify himself in these respects, and the fact that they so often fail to agree must be looked upon as evidence that we have not yet learned to use the x-ray as an instrument of precision. In the matter of rickets this conclusion takes on importance in the light of the fact that such readings as we have discussed are being quite generally accepted as decisive in research on the preventive and curative value of various foods and drugs. Considering the importance of such research and the volume of it which is carried on, it seems pertinent to inquire whether some competent authority might not undertake or promote studies which would help to clear this situation and establish standards which, if not absolute, might be more accurate and convincing than those now in vogue.

CALCIUM AND PHOSPHORUS STUDIES

XIII. The Effect of Emulsification on the Potency of Viosterol in the Treatment of Rickets in Children

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In 1931 Hess and Lewis[1] made the observation that milk rendered antirachitic by feeding irradiated yeast to cows was more potent in preventing rickets in children than either cod liver oil or viosterol containing the same number of rat units. In 1933 they[2] reported that milk made antirachitic by direct irradiation with a carbon or mercury quartz lamp was even more potent than "yeast" milk. They gave the "efficacy ratio" of irradiated milk to viosterol as 15:1, of irradiated milk to cod liver oil as 6:1, and of irradiated milk to "yeast" milk as 2:1. Although there is reason to suppose that these numerical ratios are not strictly correct, especially with respect to the relative antirachitic values of cod liver oil and viosterol, as pointed out by Shelling and Hopper,[3] and Eliot and Park,[4] there is sufficient experimental evidence to indicate that irradiated milk, or "yeast" milk, is more efficacious in preventing and curing rickets in children than are either cod liver oil or viosterol of the same potency, expressed in rat units. The reason for this difference in potency of vitamin D has so far not been adequately explained. Two possibilities suggest themselves. One, that the provitamins of milk and of commercial viosterol are quite distinct; the other, that the vehicle in which the vitamin D is administered determines its effective potency. In contrast to cod liver oil or commercial viosterol in oil, in which the vitamin D exists in large fat globules, the vitamin in milk is dispersed in much smaller globules of fat and hence is conducive to better absorption from the gastrointestinal tract. Although it is now established that vitamin D may be formed from at least eight provitamins, and that fish oils may contain a mixture of several forms of vitamin D,[5] the direct chemical determination of these differences is not an easy matter. On the other hand, the second possibility can easily be ascertained by determining the efficacy of emulsified viosterol in milk as against viosterol in oil, administered separately.

In the first experiments, a lecithin-water emulsion of viosterol, miscible in water and in milk,[6] was added to the daily formula. Later, this method of adding vitamin D to milk was abandoned, because it was discovered that the vitamin D deteriorated slowly on standing in water solution;[7] and while the deterioration can be prevented by replacing the oxygen in solution with an inert gas, this is impractical for general use.

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In subsequent experiments, therefore, the viosterol was emulsified in malt extract* or "homogenized" directly with evaporated milk, just before canning. In these preparations, the vitamin D was found to be stable, at room temperature, after one year. The malt preparation, like the lecithin emulsion, miscible in water and milk, can be added in any given amount to one or more aliquots of the daily milk formula. Each teaspoonful of the malt preparation contained 675 international units.† The evaporated milk contained 400 units per can, equal to one quart of whole milk.

The effect of the emulsification of viosterol on the healing of rickets was thus tested in three groups of children. The number of patients in each group, the age and race of each patient, the degree of rachitic involvement, the dosage of viosterol in international units, and the approximate length of time required for complete healing are given in Tables I, II, and III. The degree of rickets and the progress of healing were determined roentgenologically. In some instances, the concentrations of calcium, inorganic phosphorus, and phosphatase of the serum were determined, before and after the administration of the vitamin preparations.

TABLE I

EMULSION OF VIOSTEROL IN WATER

CASE NO.	NAME	AGE MO.	RACE	SEVERITY OF RICKETS	VITAMIN D UNITS DAILY	HEALING COMPLETE IN DAYS	COMMENT
1	H. R.	19	C	Severe	675	80	
2	M. B.	18	C	Marked	675	65	See Fig. 1.
3	L. T.	16	C	Moderately severe	4,500	37	Before therapy, Ca, 7.6; P, 1.6; after 22 days, Ca, 10.4; P, 6.6. See Fig. 2.
4	J. G.	7	C	Rickets and tetany	675	58	See Fig. 3.

RESULTS

Group I: Viosterol Emulsified in Water.—Originally, this group consisted of twelve colored children with active rickets. The first four received 675 units of the freshly prepared vitamin emulsion daily and showed complete healing within a period of two and one-half months. One of these was an eighteen-month-old colored boy who suffered from most marked rickets and osteoporosis of the entire skeleton, including the slow growing ends of the long bones (Fig. 1). A fifth patient, a sixteen-month-old boy, received daily 4,500 units of the emulsion (equal to 20 drops of viosterol) in his milk and showed complete healing within five weeks after therapy was instituted (Fig. 2). The remaining seven

*The emulsions in malt extract were prepared by the research staff of Mead Johnson and Company, Evansville, Ind.

†The potencies of viosterol throughout this paper are expressed in international units (U. S. Pharmacopeia, 1934, Rev.).

TABLE II

EMULSION OF VIOSTEROL IN MALT EXTRACT

CASE NO.	NAME	AGE MO.	RACE	SEVERITY OF RICKETS	VITAMIN D UNITS DAILY	HEALING COMPLETE IN DAYS	COMMENT
1	E. J.	29.5	C	Marked rickets and osteoporosis	675	105	See Fig. 4.
2	J. W.	13	C	Rickets, tetany, extreme osteoporosis	675	76	See Fig. 5.
3	W. P.	10	C	Severe rickets, possibly scurvy	675	150	Probably healed sooner. Mother lax in returning for x-ray examinations.
4	T. T.	7	C	Moderately severe	675	80	
5	D. H.	7	C	Moderately severe	675	75	
6	M. S.	7	C	Severe	675	80	
7	J. B.	7	Ital.	Severe	675	120	Probably healed earlier. Mother failed to bring child for more frequent x-ray examinations.

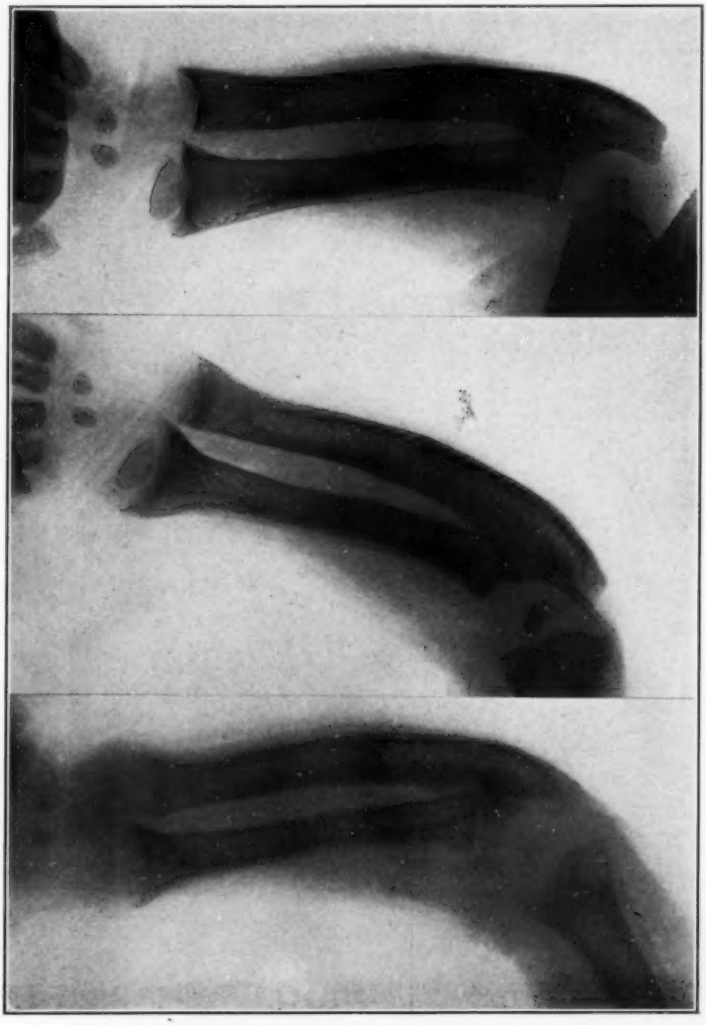
children received the 675 units of the emulsion after it had been standing in the ice box for periods of from two to five months. The roentgenograms of the bones in some of these children showed evidence of healing

TABLE III

EMULSION OF VIOSTEROL IN CANNED MILK

CASE NO.	NAME	AGE MO.	RACE	SEVERITY OF RICKETS	VITAMIN D UNITS DAILY	HEALING COMPLETE IN DAYS	COMMENT
1	M. T.	25	C	Most marked rickets and deformities of all bones	400	141	See Fig. 6.
2	J. C.	14	C	Marked rickets and osteoporosis	400	115	
3	W. T.	10.5	C	Marked rickets and osteoporosis	400	125	Sister to M. T. (Case 1). Marked rickets in several members of family.
4	H. C.	9	C	Rickets and tetany	350	86	See legend to Fig. 7, giving Ca and P findings in serum.
5	L. B.	7.5	C	Most severe rickets	400	80	
6	A. Z.	6	W	Moderately severe rickets and anemia	250-400	100	See legend to Fig. 8, giving Ca and P findings in serum.

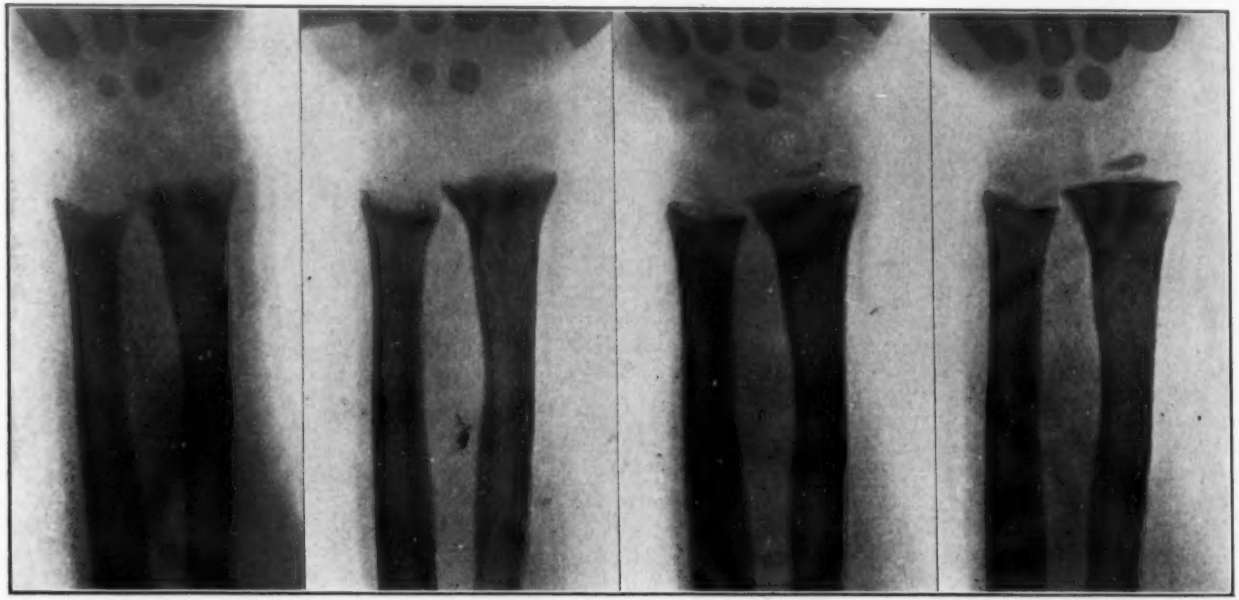
at first, but no healing after a time; others showed no healing at all, even when the dosage was increased. The failure was due, as mentioned, to the gradual deterioration of the vitamin D in the water solution. In



A. B. C.

Fig. 1.—M. B., colored male, eighteen months old. Breast fed for fifteen months. Has not walked. Said to have received one teaspoonful of cod liver oil since infancy. X-ray Diagnosis: Marked rickets and osteoporosis. Serum Ca and inorganic P, 8.0 and 3.4 mg. per cent, respectively. One week later, water-lecithin emulsion of viosterol started (675 units in milk daily). *A*, 7 days before therapy was begun, showing marked rachitic changes even at the slow growing ends; *B* and *C*, after 35 and 65 days of vitamin D therapy, respectively. The last x-ray film shows nearly complete healing.

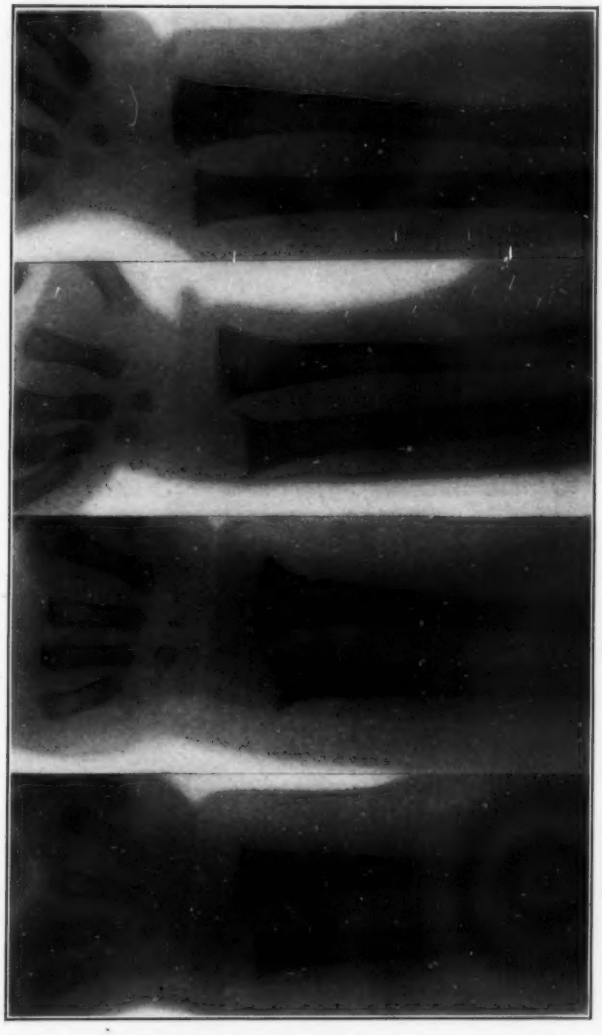
the entire group of 12 patients, therefore, complete healing of rickets was observed in only five patients, in four of whom the healing was accomplished by a daily amount of vitamin D equal to as little as 3 drops of viosterol in oil. The roentgenograms of the bones of three of the patients of this group are shown in Figs. 1, 2, and 3.



A. *B.* *C.* *D.*

Fig. 2.—L. T., colored male, aged sixteen months, received cod liver oil during early infancy; none since. X-ray Diagnosis: Moderately advanced rickets. Serum Ca and inorganic P, 7.6 and 1.6 mg. per cent, respectively. No clinical evidence of tetany, probably because of the associated low inorganic phosphorus concentration in the serum. *A*, 10 days before therapy with 4,500 units of water-lecithin emulsion of viosterol in daily milk formula; *B*, *C*, and *D*, after 9, 23, and 37 days of therapy, respectively. Serum Ca and inorganic P after 22 days of treatment, 10.4 and 6.6 mg. per cent, respectively.

Group II: Viosterol Emulsified in Malt Extract.—This group comprised six colored children and one white child of Italian parentage. Each patient received one teaspoonful of the malt emulsion in milk daily (675 units). In five of the children, the rickets healed completely in



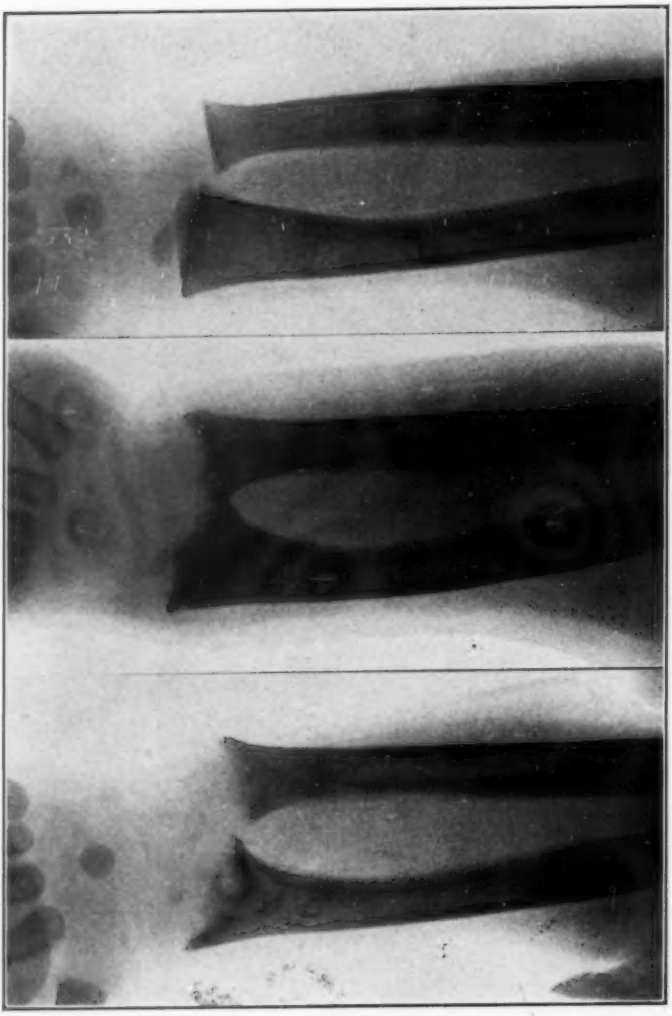
A. *B.* *C.* *D.*

Fig. 3.—J. G., colored male, aged seven months. Rickets and tetany (Chvostek sign positive). X-ray Diagnosis: Moderate rickets. Serum Ca and inorganic P, 8.8 and 3.0 mg. per cent, respectively. Treated with calcium gluconate on admission to clinic; vitamin D therapy in form of 675 units of water-lecithin emulsion of viosterol, added to milk, was started six days later. Serum Ca and inorganic P after 10 days of calcium therapy and 4 days of vitamin D administration, 9.4 and 4.9 mg. per cent, respectively. *A*, 8 days before vitamin D therapy was begun; *B*, *C*, and *D*, after 16, 44, and 58 days of treatment, respectively.

less than three months; in one, it healed in four months; and in another, in five months. The latter two probably healed sooner, but this fact could not be ascertained because of failure on the part of the mothers to bring the children for the last x-ray examination at the appointed periods. The average duration of complete healing in this group was

3.1 months. The healing brought about by this small dose of viosterol in malt extract is illustrated in Figs. 4 and 5.

Group III: Viosterol Emulsified in Canned Evaporated Milk.—This group comprised five colored children and one white Polish girl. Their



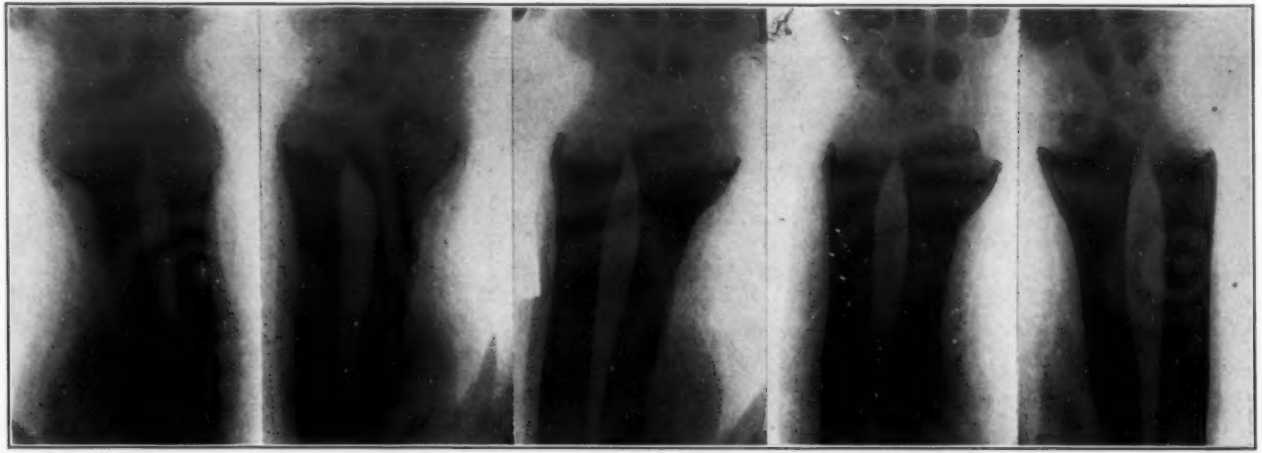
A. *B.* *C.*

Fig. 4.—E. J., colored male, aged twenty-nine and one-half months. Late rickets with deformities; definite delay in appearance of centers of ossification. Treated with 675 units of viosterol in malt extract daily, dissolved in milk. *A*, 4 days before therapy was begun; *B* and *C*, after 28 and 105 days, respectively. The amount of viosterol administered daily is equal to about 3 drops of viosterol in oil. Ordinarily, it would require at least 20 to 30 drops of viosterol, administered separately, to effect a cure in this type of rickets within a period of from four to five months.

ages ranged from six to twenty-five months. The dosage of vitamin D ranged from 240 to 400 units daily, or approximately 1.1 to 1.7 drops of viosterol in oil. One of the patients suffered from severe tetany, in addition to the rickets; two others, a brother and sister, both suffered from severe rickets. The brother showed, in addition to the marked rickets,

A. *B.* *C.* *D.* *E.*

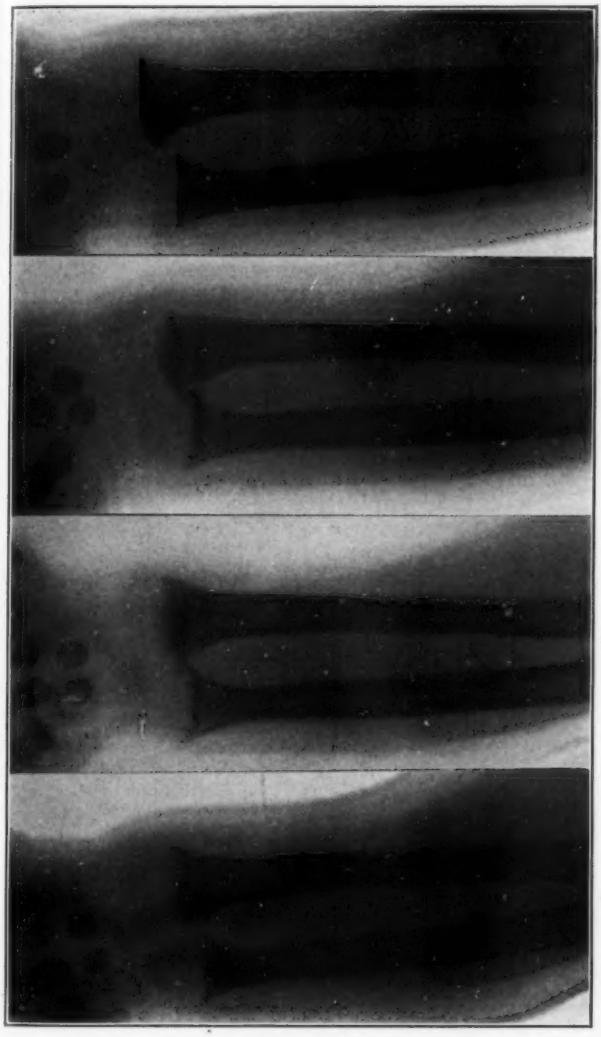
Fig. 5.—J. W., colored male, aged thirteen months. Severe rickets in all extremities, including the slow growing ends. Rickets and tetany diagnosed 45 days previously. Calcium chloride and cod liver oil were prescribed, but it is not at all certain that the latter had been given in the suggested amounts (three teaspoons daily), since the roentgenograms of the bones showed that the rickets had progressed. *A*, 6 days before therapy with malt emulsion of viosterol (675 units added to daily milk intake); *B*, *C*, *D*, and *E*, after 14, 29, 43, and 76 days of therapy, respectively.



A. *B.* *C.* *D.* *E.*

Fig. 6.—M. T., colored male, aged twenty-five months. Marked rickets, osteoporosis, and deformities in several members of the family. A younger sister, ten and a half months old, treated at the same time, showed complete healing within four months after therapy with viosterol milk was instituted. *A*, 17 days before therapy with canned vitamin D milk begun (400 units daily, equal to 1.7 drops of viosterol in oil). *A*, x-ray of the long bones taken two days before therapy was instituted showed no healing. *B*, *C*, *D*, and *E*, after 56, 86, 113, and 141 days, respectively. Considering the severity of the rickets and the age of the child, it is remarkable that healing took place with this small amount of viosterol in this comparatively short time. Ordinarily, it would require 40 to 60 drops of viosterol, administered separately, to bring about this degree of healing.

general osteoporosis and deformities of nearly the entire skeletal system, including the calverium. He received daily one can of the milk, containing 400 units of viosterol, and showed complete healing in less than



A. B. C. D.

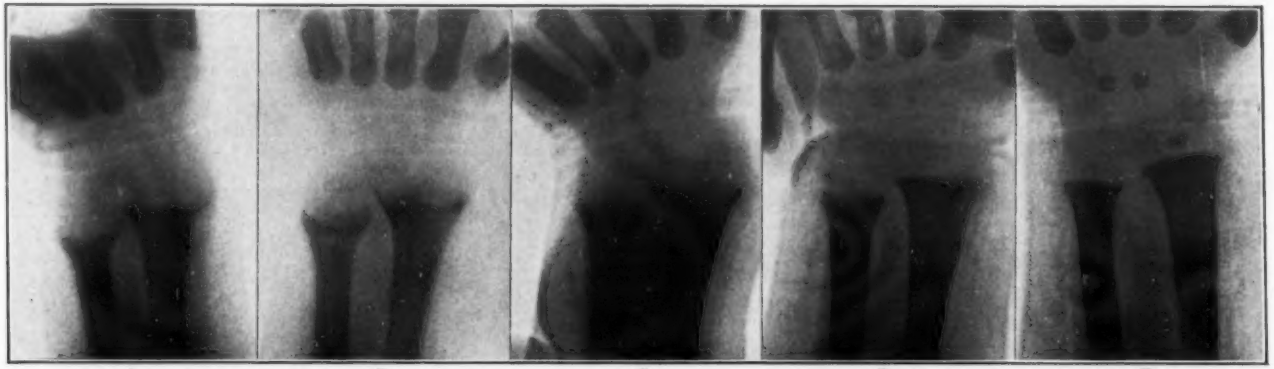
Fig. 7.—H. C., colored male, aged nine months, was admitted to the hospital because of convulsions and difficulty in breathing. Diagnosis: Rickets, tetany, and pneumonia. Serum Ca and inorganic P, 6.3 and 3.6 mg. per cent, respectively. Ca gluconate and chloride without vitamin D were given for 17 days. The acute symptoms of tetany subsided, but the X-ray examination of the bones showed progression in the rachitic process. *A*, 4 days before therapy with canned viosterol milk was started (350 units daily); *B*, *C*, and *D*, after 25, 55, and 86 days of therapy, respectively.

CHEMICAL FINDINGS IN THE SERUM

DAYS IN HOSPITAL	CA MG. %	P MG. %	PHOSPHATASE*
on admission	6.3	3.6	11.4
3 days	7.2	3.8	17.5
8 days	7.9	4.1	21.9
17 days†	8.0	3.5	28.6
48 days	9.8	5.2	17.3
92 days	10.6	6.1	17.0

*Bodansky units.
†Viosterol milk begun and continued for four months.

five months (141 days). Considering the degree of rickets present and the small dosage of vitamin D, it is remarkable, indeed, that healing took place in this child within this period of time (Fig. 6). The sister, in whom the rachitic process was also quite marked, received the same amount of canned viosterol milk and showed complete healing of the



A. *B.* *C.* *D.* *E.*

Fig. 8.—A. Z., white female, six months old, admitted to the hospital because of poor nutrition, anemia, rickets, and possibly scurvy. Remained in the metabolism ward of hospital for more than 8 months. Roentgenograms of the extremities taken weekly except for the last month's stay in the hospital. She was fed a whole powdered milk formula exclusively for a preliminary period of 6.5 weeks, after which time she was given canned viosterol milk, beginning with 240 units and increasing gradually to 400 units of vitamin D daily. *A*, at beginning of vitamin D therapy; *B*, *C*, and *D*, after 22, 50 and 85 days of therapy, respectively. The roentgenogram, *E*, was taken one month after the patient was discharged from the hospital. Since the roentgenogram, *D*, shows nearly complete healing, it is reasonable to assume that the rickets healed completely in the period between the taking of the last two roentgenograms. Complete healing probably occurred within three or three and a half months.

CHEMICAL FINDINGS IN THE SERUM

	CA	P
2/16/35	8.6	4.0
2/25/35	–	4.0
3/ 4/35*	8.4	4.2
5/16/35	9.3	5.6

*Viosterol milk started 3/18/35.

rickets within four months. The remaining four patients healed within an average period of three months. The patient suffering from tetany was observed in the hospital for a period of thirty-five days; and the white child remained in the hospital, as a subject of metabolic study, for more than eight months. In both of these patients, it was thus possible to obtain preliminary observations before therapy with vitamin D milk was started and to observe the chemical changes in the blood and the roentgenologic changes in the bones at frequent intervals. The colored patient with rickets and tetany received 350 units of vitamin D in his daily milk formula and showed complete healing of the rachitic process in less than three months. The white child, because of her age and smaller caloric intake, ingested only 240 units of vitamin D daily; but, as the caloric intake was increased, the dosage was increased to 400 units daily. In this patient, healing was noted in the x-ray films within twelve days and complete healing after three months of therapy. The effects of these small amounts of vitamin D emulsified in milk on the healing of severe rickets are illustrated in Figs. 6, 7, and 8.

COMMENT

The results of the above study with emulsified vitamin D in the cure of rickets in children are in accord with what Lewis[8] found in his studies with irradiated ergosterol dissolved in propylene glycol. This preparation, like the emulsions of vitamin D used in the present study, is miscible in milk; and, when administered in this vehicle, is able to prevent or cure rickets with much smaller doses than could be accomplished by either cod liver oil or by viosterol in oil. The curative doses in the present study, and both the curative and preventative doses employed by Lewis, seem to be as effective as irradiated or "yeast" vitamin D milk containing approximately the same number of rat units. This suggests the possibility that the greater potency of vitamin D in milk rendered antirachitic by various means, such as direct irradiation, feeding vitamin D to the cow, or by the addition to milk of crystalline vitamin D in propylene glycol or of emulsified viosterol in oil, is due to the small size of the particles of the lipoids carrying the vitamin. For this reason, it is probably absorbed to a greater degree from the gastrointestinal tract. In the emulsions of viosterol, this reduction in size of the lipoids is accomplished mechanically;* in milk rendered antirachitic by feeding vitamin D to the cow, the emulsification occurs *in vivo*, that is, the fat globules are naturally small; in milk to which is added irradiated ergosterol in propylene glycol, the vitamin becomes dispersed within the small fat globules; and in milk irradiated directly, the same small particles of fat, containing the provitamin, acquire antirachitic properties.

Although discrepancies still exist between the antirachitic efficacy of vitamin D found in fish livers and that produced by irradiation of sterols,

*The particle size of the water-lecithin emulsion was usually smaller than a human red blood cell.

these differences are most pronounced in chickens, and less so in rats and human beings. In human beings, the discrepancy has been nearly abolished by more controlled clinical studies, showing that, unit per unit, viosterol is as efficacious as cod liver oil in preventing and curing rickets. The present study with emulsions of viosterol in oil and the investigations of Lewis with crystalline vitamin D in propylene glycol indicate that vitamin D from various sources may possess equal therapeutic value in children, provided the physical state of the vitamin is the same, or nearly the same, in all cases. Thus, when the results of studies with vitamin D added to milk are compared to the antirachitic value of irradiated or "yeast" milk, it becomes evident that, within the limits of bio-assay and individual differences in children, the therapeutic ratios between these various forms of vitamin D milks approach unity. The studies with the various kinds of vitamin D milk, including milk rendered antirachitic by the addition of cod liver oil concentrates, suggest that the enhancement of the antirachitic potency of vitamin D is due to the reduction in size of the lipoids carrying the vitamin. The suggestion, made by Jeans,[10] that "it would not be surprising to find that all vitamin D of animal origin has a similar potency and all vitamin D of vegetable origin another potency" is not borne out by the observation of Bills[5] that even fish oils show discrepancies in potency between the rat and the chicken. It is also rendered improbable by the fact that, in human beings, the antirachitic ratio between viosterol milk and milk rendered antirachitic by the addition of cod liver oil concentrate approaches equality.

The degree of enhancement of the antirachitic potency of vitamin D by emulsification may be approximated by comparing the results obtained in the present study with that made previously on a larger group of rachitic children, treated with viosterol, administered alone.[3] In the latter group, comprising 164 rachitic children, the average dose of viosterol required to heal all degrees of rickets within a reasonable time was 20 drops, or about 4,500 international units, and patients with very severe rickets required as much as two to three times that amount. Attempts to cure moderate or severe degrees of rickets with 2, 3, 5, or even 10 drops of viosterol were invariably unsuccessful, the roentgenograms of the bones showing only very slight healing after some time or, with the smaller dosages, a progression of the disease. Therefore, the complete healing brought about in patients with moderate and severe degrees of rickets with as little as 240 to 675 units of vitamin D, in the form of viosterol emulsified in milk, suggests that the antirachitic potency was enhanced approximately tenfold by reducing the particle size of the fat in which the vitamin D is carried.*

*These studies were made during the winters of 1933-34 and 1934-35. Since this paper was originally prepared for publication, Eliot and her associates[9] reported that 400 units of viosterol homogenized in evaporated milk prevented rickets in most of the twenty-nine white and twenty-one colored infants.

The discrepancy between the potency of vitamin D milk, as determined in the rat, and its efficacy in human beings may be explained by the fact that when such milk, or milk fat, is tested for its antirachitic potency in rats, it is usually mixed with a dry cereal diet. This procedure renders the particles of lipoid no longer in a state of emulsion, and hence no longer as readily absorbable as when they are contained in milk and fed directly as such to human beings.

Attempts to produce liquid rachitogenic diets to which emulsions of vitamin D could be added, in order to thus prove the greater absorbability of vitamin D in the emulsified form from the gastrointestinal canal, culminated in failures. On the ordinary Steenbock and Black rachitogenic diets, intraperitoneal injections into rats of the water-lecithin emulsion of viosterol proved to be more efficacious in preventing and curing rickets than a similar amount of viosterol fed along with the diet. This may be interpreted to mean that the vitamin is absorbed more readily from the peritoneal cavity, but it does not prove that a similar process takes place in the gastrointestinal canal. In the absence of definite proof, the suggestion that the enhancement of vitamin D is due to greater absorption from the gastrointestinal tract must be considered as tentative.

The addition of viosterol to milk offers a practical and cheap means of providing vitamin D in more effective amounts than is possible by direct irradiation or by feeding vitamin D to the cow.

SUMMARY

The addition of viosterol to milk in the form of an emulsion enhances the efficacy of the viosterol, in curing moderate and severe degrees of rickets, about tenfold. It is suggested that the increased potency of milk rendered antirachitic by direct irradiation, by feeding vitamin D to the cow, or by the addition of vitamin D which ultimately becomes dispersed within the fat globules, is due to the small particle size of the lipoids in which the vitamin is carried, and hence is probably more easily absorbed from the gastrointestinal canal.

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555 Prospect Place

THE COMPOSITION OF AN AVERAGE PEDIATRIC PRACTICE

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WHAT type of case will be most frequently encountered when practice is begun?" Aldrich[1] and Arena and Harris[2] have described the composition of suburban, hospital, and dispensary practice, and, because no one seems to have recorded the diseases of the mixed group of children from urban, suburban, rural, and industrial districts, a system of records* was designed to gather this information. The diseases tabulated below represent those in the first 1,500 children under my care. The figures represent the number of children in which each disease has occurred and not the number of times the disease has occurred. Of course, many of these children had more than one of these different diseases.

TABLE I

NEWBORN AND CONGENITAL DEFECTS		TOTAL 129 OR 8.60%	
Prematurity	28	Absence of vocal cords	1
Hematoma, sternomastoid	3	Accessory ears	1
Cephalhematoma	1	Supernumerary toe	1
Fractured clavicle	1	Redundant sigmoid	1
Intracranial hemorrhage	3	Imperforate anus	1
Atelectasis	6	Atresia of esophagus	1
Omphalitis	1	Hernia of umbilical cord	1
Tetany of the newborn	2	Hypospadia	1
Congenital syphilis	7	Exstrophy of bladder	1
Cyst of lip	2	Encephalocele	1
Cyst of floor of mouth	2	Spastic paraplegia	8
Harelip	2	Thymus hypertrophy	6
Central scotoma	1	Amyotonia congenita	1
Dermoid of cornea	1	Congenital heart	7
Tongue-tie	4	Hemangioma	8
Phimosis	10	Umbilical hernia	15

All infants weighing less than 5 pounds and who were unable to maintain a normal body temperature under routine newborn care were classified as premature. All of these infants were fed evaporated milk (unsweetened); occasionally lactic acid drops were added to the formula. These children were kept in warmed beds in the ordinary newborn nursery. It is interesting to note that atelectasis was diagnosed twice as frequently as intracranial hemorrhage, and I believe

*The history, examination, and progress notes are taken in an abbreviated form and then dictated to the stenographer, who types them on a 4 by 6 inch card. This card is kept in an envelope on the outside of which is written the name of the patient. In one corner under the patient's name are listed the diagnoses which have been made in that patient. On the opposite corner is printed a list of the immunizations to be done, and, as these are given, they are checked off. With this method, a glance at the patient's card shows the diseases he has had and the immunizations which have been done or should be done. This card is filed alphabetically under the patient's name. A separate diagnosis file is kept according to diseases, and the child's name is entered if he contracts a disease. A check is placed beside the diagnosis on the patient's card when that diagnosis is entered on the index card. If a patient has a recurrence of the same disease, it is not entered again.

that if the infants who do badly immediately after birth were more thoroughly examined, fewer diagnoses of intracranial hemorrhage would be made. The spastic paraplegias listed may have been due to intracranial hemorrhage, yet a review of the neonatal and early infancy period of these children does not reveal any symptoms suggesting that condition. The thymus hypertrophy patients all had periods of cyanosis and an enlarged substernal shadow, both of which disappeared after x-ray therapy.

TABLE II

ROUTINE CARE		TOTAL 469 OR 31.26%	
Feeding regulation	403	Adherent foreskin	15
Appetite cultivation	29	Adhesions of labia	2
Colic	4	Rickets	7
Thrush	9		

Evaporated milk has been used routinely in feeding all normal babies unless the parents objected to canned food. No attempt has been made to include the diagnoses, behavior problems, and psychologic maladjustments in the diagnoses lists because in practically all patients there is some behavior problem or some minor psychologic maladjustment which requires the thoughtful attention of the pediatrician.

TABLE III

IMMUNIZATIONS		TOTAL 604 OR 40.26%	
Diphtheria	217	Smallpox	70
Schick test	129	Typhoid	78
Pertussis	89	Measles	14
Poliomyelitis	7		

The relatively small number of immunizations is not a reflection upon the diligence of the physician nor the willingness of the parents to have the children immunized, but rather an index to the activity of the local health department. Diphtheria immunization is advised in all children six months of age or over, and a majority of the 403 children listed as feeding regulation cases have been immunized. Those not listed here have received diphtheria toxoid at the health department free of charge. Many parents insist upon waiting for typhoid and smallpox immunization until the child is of preschool age, and having these immunizations given free at the preschool clinic. Others, when immunization is suggested, frequently state that they will go to the health department where this can be obtained free of charge. Since the local health department has not as yet undertaken the task of whooping cough immunization, the figures show that more children were immunized against pertussis in a period of four months than were given typhoid or smallpox vaccine over a period of four years. Convalescent serum was given to the patients listed as having measles and poliomyelitis immunization.

TABLE IV

GASTROINTESTINAL DISORDERS		TOTAL 236 OR 15.73%	
Diarrhea	109	Glossitis	2
Bacillary dysentery	24	Gastritis	12
Acute intestinal intoxication	1	Appendicitis	9
Amebic dysentery	1	Meckel's diverticulum	1
Pylorospasm	15	Impacted feces	1
Pyloric stenosis	5	Rectal polyps	4
Catarrhal jaundice	5	Rectal prolapse	1
Cardiospasm	2	Constipation	12
Duodenal spasm	1	Hemorrhoid	1
Spastic colon	10	Hypoglycemia	1
Mesenteric adenitis	6	Intestinal influenza	13

All children with stools sufficiently loose to demand an office or home visit, other than those with bacillary dysentery, have been included under the term "diarrhea." No attempt has been made to classify them as infectious, dietary, parenteral diarrhea, or ileocolitis. I was surprised to find the incidence so low. Of the 109 patients, the condition was sufficiently severe in 22 to require hospitalization. In only one patient was mastoidectomy done, and in this patient the diarrhea had ceased and the operation was done because of the chronic draining ear and not for the diarrhea. Most of the bacillary dysentery occurred during one summer season and all came from rural districts. Four of five patients who had pyloric stenosis were operated upon (Table XVI). A definite tumor was found in each, and immediate relief followed the operation. The fifth patient was not operated because of the parents' objections and finally recovered, but not without a very stormy course. It is worthy of note, because of the scarcity of such reports in the literature, that one of the patients operated upon was a negro infant and the tumor found was the largest of this series. The symptom-complex leading to the diagnosis of spastic colon included a feeling of fullness after the ingestion of a small amount of food, abdominal pain, especially during or after a meal, the passage of hard, lumpy stools, and x-ray evidence of spasticity of the colon. Headaches, lassitude, coated tongue, and foul breath were common complaints. The symptoms and signs responded well to a bland diet, mineral oil, and belladonna. Children suffering from mesenteric adenitis have gastric symptoms similar to those with spastic colon. In addition there is a history of repeated attacks simulating appendicitis, yet all of the signs of appendicitis are not present. These children usually have had repeated upper respiratory infections. Abdominal examination fails to reveal anything characteristic, but rectal examination frequently reveals the presence of enlarged glands. The diagnosis of intestinal influenza is presented with the full realization of its questionableness. However, the symptoms so classified occur chiefly at the time of year when respiratory diseases are most prevalent

and especially during an influenza epidemic. Frequently other children in the family will have the respiratory symptoms of influenza at the same time.

TABLE V

ACUTE INFECTIONS OF THE UPPER RESPIRATORY TRACT	TOTAL 656 OR 43.73%
Upper respiratory infections (including rhinitis, pharyngitis, tonsillitis, catarrhal otitis)	482
Otitis media, purulent	87
Otitis externa	3
Adenitis, cervical	27
Laryngitis, streptococcic	1
Conjunctivitis	16
Sinus infection	14
Bronchosinusitis	10
Retropharyngeal abscess	2
Peritonsillar abscess	1
Croup	13

This respiratory group constitutes the bulk of pediatric illness. Four hundred and eighty-two of the 1,500 children were seen at least once with upper respiratory infections and many of them had them repeatedly. Only those patients in whom paracentesis was done or in whom the tympanic membrane ruptured spontaneously were classified as having purulent otitis media. It is of interest that mastoiditis occurred in only one of these, an infant of four months. The remaining cases of mastoiditis (Table XVI) were referred by other physicians. Only cases of epidemic conjunctivitis are listed here; those occurring during an upper respiratory infection are included under that diagnosis. Bronchosinusitis is used to describe the children who come in with the chief complaint of chronic cough. Examination of these patients reveals coarse râles at the bases posteriorly, congested nasal mucous membrane and a postnasal drip. A roentgenogram of the sinuses discloses evidence of sinusitis, and the cough promptly disappears following treatment of the sinus infection.

TABLE VI

ACUTE PULMONARY INFECTIONS	TOTAL 222 OR 14.80%
Pneumonia	76
Empyema	10
Bronchitis	23
Influenza	79
Pleurodynia	14
Bronchiectasis	4
Hilum tuberculosis	14
Pulmonary tuberculosis	1
Lung abscess	1

The diagnosis, pneumonia, given here includes both lobar and bronchopneumonia. No case was so diagnosed unless there were definite signs of consolidation, on physical examination or by x-ray. Thirty-six of these patients were treated in the hospital; the remaining ones were treated at home. The only therapy other than symptomatic

treatment has been blood transfusions. The benefit of this procedure has been repeatedly reported. Several of the empyema patients were referred and were classified as such, and not as pneumonia with empyema, which accounts for the apparently high incidence. The children recorded as having bronchitis had fever, cough, and a variety of râles in the chest. Some of these probably should be classified as pneumonia. The patients with cough and scattered râles in the chest following upper respiratory infections are included in that diagnosis. The cases of pleurodynia all occurred during one epidemic and were quite alarming at the onset. The children presented a picture of either toxic pneumonia or ruptured gastric ulcer, depending upon whether the pain was in the chest or the abdomen. The treatment was symptomatic and all of this series recovered spontaneously. The bronchiectasis patients have done well with postural drainage and elimination of foci of infection in the upper respiratory tract. Bronchoscopic treatment was not resorted to in any of these. The one case of lung abscess was due to Vincent's organisms and responded well to arsphenamine therapy.

TABLE VII

ACUTE INFECTIOUS DISEASES		TOTAL 278 OR 18.53%	
Measles	38	Poliomyelitis	3
Mumps	11	Rheumatic fever	2
Chickenpox	25	Chorea	1
Pertussis	86	Erysipelas	6
Scarlet fever	12	Vincent's infections	22
German measles	11	Septicemia, streptococcic	4
Diphtheria	17	Septicemia, staphylococcic	1
Roseola infantum	27	Tetanus	2
Typhoid	8	Rocky Mountain spotted fever	2

Most of the mumps and chickenpox patients were cared for by the parents, and the physician was simply notified that the disease had occurred. Record of this was made on the child's card, but only those who were seen were placed on the diagnosis file. The relatively few cases of measles seen is again accounted for by the activity of the health nurses who diagnose the cases, placard the house, and suggest therapeutic procedures. The number of cases given here is the number actually seen. Whooping cough cases oftentimes become a little too difficult for the health nurse; thus the number of cases of whooping cough seen is more than double the figure for measles. Most of the cases of diphtheria occurred in rural districts. Only those patients with membranes and positive cultures were so diagnosed. Six of these were laryngeal cases requiring either intubation or tracheotomy. The cases of Rocky Mountain spotted fever occurred in a rural district. The two children lived within a mile of each other, but their illnesses occurred a year apart.

The 52 cases listed as routine examination were children in whom no abnormality was found. Those designated as fever of undetermined origin represent a group of children in whom the mothers happened

TABLE VIII

ROUTINE EXAMINATION AND MISCELLANEOUS	TOTAL 430 OR 28.66%
Postural defects	12
Tonsil and adenoid abnormalities	98
Functional heart murmur	15
Flatfoot	4
Knock knees	7
Dental caries	55
Bow legs	3
Routine examinations	52
Tuberculin tests	118
Fever, undetermined origin	5
Headache	11
Intestinal parasites	23
Poisoning, chemical	9
Torticollis	2
Pneumococcic peritonitis	1
Purpura hemorrhagica	1
Gangrene, penis, leg, arm	1
Tetany	2
Scurvy	4
Malaria	5
Hysteria	1
Drug addiction	1

to find an elevation of temperature and then by repeated examinations found this temperature to be present over a long period of time. The temperature usually ranged between 37.8° and 38.2° C. (100.2° and 100.6° F.). These children presented negative physical examinations and were in most cases of normal weight; all laboratory tests were negative. The therapy in these cases consisted of getting rid of the thermometer. The one case listed as purpura hemorrhagica was secondary to malaria. The case of gangrene of the penis, legs, and arms was an extremely interesting one and will be reported elsewhere. The condition followed an infection of the mouth and throat with a *Staphylococcus hemolyticus*. The one case of drug addiction occurred in an infant six months old and was due to a soothing syrup, which contained tincture of opium. The infant was placed in the hospital in order to break the habit but because of his continuous crying the parents took it home where the supply of cordial was plentiful. It has not been seen since.

TABLE IX

ALLERGIC MANIFESTATIONS		TOTAL 99 OR 6.60%	
Asthma	35	Hay fever	3
Eczema	45	Abdominal allergy	2
Urticaria	12	Dermographia	2

The high incidence of asthma is in accord with the observation of other physicians in the community. It has been suggested that the contamination of the air with tobacco dust might account for this; yet very few of these patients show any skin sensitivity to tobacco extract. Serum sickness has not been recorded as it has been considered an expected sequela to serum therapy and not a compiication. All cases of eczema, from the infantile facial type to the severe general type, are

included. In this treatment, skin tests, with the elimination of the positive reactors, have been disappointing. Most of the mild cases have cleared up with the application of the usual tar preparations. The more severe ones have shown temporary improvement with applications to the skin, and all eventually have cleared up with the passage of time.

TABLE X

ACCIDENTS		TOTAL 63 OR 4.20%	
Miscellaneous injuries	25	Dog bites	9
Burns	12	Sprains	8
Concussion of brain	2	Swallowed foreign body	4
Skull fracture	3		

Miscellaneous injuries included lacerations and bruises not requiring major surgical procedures. None of the children bitten by dogs were given antirabic treatment.

TABLE XI

DISORDERS OF THE NERVOUS SYSTEM		TOTAL 108 OR 7.20%		
Anorexia	29	Erb's palsy		1
Enuresis	5	Bell's palsy		1
Convulsions	16	Meningismus		1
Stammering	2	Encephalitis		5
Epilepsy, grand mal	11	Meningitis		19
Epilepsy, petit mal	2	Meningococcic	3	
Hysteria	1	Pneumococcic	3	
Pavor nocturnus	1	Influenzal	2	
Somnambulism	1	*Bacillus coli*	1	
Masturbation	3	Tuberculous	7	
Idiocy, unclassified	5	Mumps	1	
Hydrocephalus	3	Aseptic	2	
Microcephalic idiocy	2			

The unclassified convulsion cases represent those children who have convulsions with the onset of infectious diseases and other disturbances and who do not show any evidence of organic disease of the nervous system. Therapy of epilepsy has been extremely disappointing, and none of these patients has been permanently relieved by any of the therapeutic procedures tried, although ketogenic diets, dehydration therapy, luminal, and bromides have all been given a trial.

The comparatively large number of cutaneous lesions suggests the advisability of including more time for the treatment of skin diseases in our medical school curriculums.

Two of the patients with hyperthyroidism were adolescent, and the condition subsided after this period was past. The third patient was a child of ten years who presented early toxic symptoms. She was given x-ray therapy over the thyroid, and these symptoms subsided. The one patient who had goiter was a four-year-old negro girl, who moved from town and nothing is known of her further progress.

The one case of myocarditis resulted from diphtheria. The anemia recorded was of the secondary variety, either as the result of infection

TABLE XII

CUTANEOUS LESIONS		TOTAL 243 OR 16.2%	
Local infections	27	Ingrowing toenail	7
Toxic erythema	7	Cellulitis	2
Dermatitis	3	Straw itch	1
Ringworm	5	Vitiligo	1
Poison ivy	12	Intertrigo	12
Impetigo	57	Seborrheic dermatitis	11
Insect bites	13	Molluscum contagiosum	1
Pityriasis rosea	1	Papular urticaria	3
Scabies	16	Lichen urticatus	3
Paronychia	8	Urticaria pigmentosa	1
Stye	8	Granuloma annulare	1
Furunculosis	5	Erythema multiforme	1
Herpes zoster	2	Heat rash	24
Acne	11		

TABLE XIII

NUTRITIONAL AND GLANDULAR CONDITIONS		TOTAL 41 OR 2.73%	
Overweight, low B.M.R.	7	Hypertension, adolescent	2
Fröhlich's syndrome	2	Hyperthyroidism	3
Malnutrition	9	Goiter	1
Adenitis, not cervical	11	Cretin	2
Diabetes	3	Small stature	1

TABLE XIV

CHRONIC CONDITIONS		TOTAL 43 OR 2.86%	
Endocarditis	3	Arthritis	1
Myocarditis	1	Hypoglycemia	1
Anemia	35	Argyria	2

or inadequate nutrition. The two patients who had argyria moved to our community from other sections of the country, and the argyria was present when they were first seen. There was a history of the use of colloidal silver preparations in each case.

TABLE XV

GENITOURINARY DISORDERS		TOTAL 92 OR 6.13%	
Pyelitis	58	Vaginitis, nonspecific	4
Nephritis	12	Vaginitis, gonorrheal	5
Hydrocele	12	Menorrhagia	1

The incidence of pyelitis seems rather high, yet no case was so diagnosed except in the presence of fever and many pus cells in clumps in the urine. All of these cases were in girls. All except one of the nephritis cases were of the acute hemorrhagic variety; four followed impetigo; the remaining ones were secondary to upper respiratory infections. Two of these patients showed cerebral manifestations which were controlled by magnesium sulphate given intramuscularly.

The figures given in this table represent patients operated upon except for one of the infants who had pyloric stenosis, and seven of the inguinal hernia patients.

TABLE XVI

SURGICAL CONDITIONS		TOTAL 117 OR 7.80%	
Acute appendicitis	5	Fractures	7
Peritonitis	2	Osteomyelitis	3
Intussusception	2	Tonsillectomies	71
Meckel's diverticulitis	1	Mastoiditis	6
Intestinal obstruction	2	Antrum drainage	1
Pyloric stenosis	5	Inguinal hernia	10
Ruptured liver	1	Ruptured spleen	1

TABLE XVII

DEATHS		TOTAL 38 OR 2.53%	
Prematurity	6	Meningitis	
Congenital atelectasis	1	Epidemic	1
Congenital syphilis	1	Influenzal	3
Congenital atresia of esophagus	1	Pneumococcic	1
Encephalocele	1	Streptococcic	1
Intracranial hemorrhage	1	*Bacillus coli*	1
Brain tumor	1	Tuberculous	6
Pneumonia	4	Tetanus	1
Pneumococcic peritonitis	1	Bacillary dysentery	1
Septicemia, streptococcic	1	Diarrhea with acidosis	1
Septicemia, staphylococcic	1	Mastoiditis	1
Septicemia, pneumococcic	1	Marasmus	1

The high death rate in premature infants is partially accounted for by the loss of three at one time as the result of a respiratory infection which invaded our nursery for newborn infants. The premature infants are now kept in a special room which is strictly isolated, and we hope that this will not recur. The one mastoiditis patient was sent to the hospital moribund and died within a few hours of admission. The death from epidemic meningitis occurred in a three-month-old infant. The deaths from bacillary dysentery and diarrhea and acidosis occurred in referred patients within twenty-four hours of admission to the hospital.

TABLE XVIII

SUMMARY	TOTAL 3830	
Newborn and congenital defects	129	3.37%
Routine care	469	12.24%
Immunizations	604	15.61%
Routine examinations and miscellaneous	430	11.11%
Acute upper respiratory infections	656	16.96%
Acute pulmonary infections	222	5.74%
Gastrointestinal disorders	236	6.10%
Acute infectious diseases	278	7.16%
Accidents	63	1.37%
Allergic conditions	99	2.56%
Chronic conditions	43	1.11%
Nutritional and glandular conditions	41	1.06%
Disorders of the nervous system	108	2.72%
Cutaneous lesions	243	6.23%
Genitourinary disorders	92	2.37%
Surgical conditions	117	3.02%

Only one of the children who died had been seen prior to the onset of the disease causing the death. This patient, who died of pneumo-

coccic peritonitis, had been seen in the public feeding clinic. The child with staphylococcic septicemia was twelve days old. The focus of infection was an abscessed tooth which was erupted at birth.

The variety of diagnoses made in the first 1,500 children seen in private practice is summarized in this Table XVIII. The figures indicate that routine care, prophylaxis, and upper respiratory diseases make up the bulk of pediatric practice.

TABLE XIX

THE DISTRIBUTION OF CHILDREN'S DISEASES IN HOSPITAL DISPENSARY AND PRIVATE PRACTICE

GROUP OF DISEASES	HOSPITAL AND DISPENSARY BALTIMORE, MD. (147,359 CASES) (%)[2]	PRIVATE PRACTICE DURHAM, N. C. (3,224 CASES) (%)
I. Respiratory (including nose, throat, and ear conditions)	35.2	37.7
II. Gastrointestinal (including mouth, nutritional, deficiency and abdominal except kidney and spleen conditions)	26.3	28.5
III. Skin and contagious diseases (exanthems) (6 per cent in adults[3])	16.5	12.9
IV. Neuropsychiatric (including diseases of the eye)	10.1	4.0
V. Circulatory, metabolic and glandular (including heart, cardiovascular, blood, spleen, reticuloendothelial, endocrine and lymph node conditions)	4.1	3.9
VI. Genitourinary (urologic) conditions	3.4	3.8
VII. Bone, muscle, cartilage, spine and joint (orthopedic) conditions	1.7	3.1
VIII. Unclassified	2.7	6.1

COMMENT

A reclassification of the cases according to the eight groups brings out clearly the similarity of hospital and dispensary practice to private practice and indicates that a sufficient variety of cases are seen in the hospital and dispensary to equip a physician for private practice. The high incidence of skin diseases in children in both hospital and private practice as compared with the incidence in adults (6 per cent[3]) emphasizes the necessity for more complete instruction in skin diseases for a physician who is preparing to practice pediatrics. The preponderance of neuropsychiatric cases in the hospital (10.1 per cent) as compared with private practice (4 per cent) is accounted for by the large number of idiots, epileptics, and other mental defectives referred to medical centers.

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ROENTGEN RAYS IN THE TREATMENT OF ACUTE CERVICAL ADENITIS

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ENLARGEMENT of the cervical lymph nodes accompanying upper respiratory infections is particularly common in infants and children. Termination in suppuration is rather frequent,[1] and this outcome is apparently uninfluenced by local applications or other medical measures now employed. Roentgen therapy would appear to be a most desirable form of treatment since it is relatively easy and safe for a competent radiologist to administer, and yields rapid results in acute inflammatory conditions.

Despite a voluminous literature on the value of roentgen therapy in inflammatory diseases, only two papers[2, 3] are devoted exclusively to its use in acute cervical adenitis. However, many reports[4] contain uniformly successful results in approximately 80 per cent of all cases of acute lymphadenitis. This encouraged us to attempt to evaluate this mode of treatment. We wish to record our results with x-rays in a controlled series of hospitalized cases and compare them with other measures employed in the treatment of acute cervical adenitis. No previous report of a similar nature has come to our attention.

Alternate cases admitted to the pediatric ward were selected for radiation as the only form of local therapy. The others were treated by the usual medical procedures (compresses and ointments), and served as controls. General nutritional measures were the same in both groups. Only patients with marked glandular swelling, potentially suppurative, were included in this study. The symptomatology included fever (100° F. or over), local pain at the site of the swollen glands, anorexia, sleeplessness, irritability and occasionally neck stiffness. Usually there was an accompanying pharyngitis, tonsillitis, or otitis. Staphylococcus and streptococcus were the organisms cultured from the throat and suppurative glands. The roentgen treated cases slightly outnumber the controls because toward the termination of the study, since x-ray treatment had proved definitely superior to other therapeutic measures, all patients were treated by radiation.

TECHNIC*

Roentgen therapy was commenced as soon as possible after the diagnosis was established. The following factors were used in obtain-

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ing the x-rays: 125 kilovolts peak current, 25 cm. target-skin distance, with a filter of ¼ mm. copper and 1 mm. aluminum. The treatment consisted of about 80 r (measured in air without backscatter) delivered to a field about 10 cm. in diameter centered directly over the enlarged nodes. This dose is between 10 and 20 per cent of the skin erythema dose. When there was bilateral involvement, each side was exposed on successive days. If suppuration seemed imminent, only 40 to 60 r were used as the initial dose because it was believed that larger amounts might cause too sudden and extensive necrosis. Irradiation was repeated every three to five days until a satisfactory result, as judged by decreased swelling and constitutional symptoms, was obtained.

DISCUSSION

This series comprises 62 roentgen ray treated patients with acute cervical adenitis, 21 of whom were hospitalized at the San Francisco Hospital throughout the entire course of the illness. The remainder were treated in the out-patient clinic of the University of California Hospital. In Table I is a synopsis of the results in the hospitalized irradiated cases, while Table II contains similar data for the control group. Table III summarizes these findings. Of the 21 hospital patients treated with roentgen rays, 16 were males. The ages ranged from eighteen months to thirteen years. In the majority of cases the condition was secondary to an acute upper respiratory infection. The duration of the adenitis before therapy was instituted ranged from 2 to 28 days, with an average of 8.3 days. The number of treatments ranged from 1 to 5: 5 patients received 1 treatment; 7 had two; 7 others three, and the remaining 2 had four and five, respectively.

Most authors agree that a few exposures are usually sufficient to cause practically entire eradication of the condition. Indeed, Desjardins[5] has stated that "if the inflammatory process does not respond to three separate irradiations it is not likely to yield at all." This has been our experience also.

As high as 200 or more roentgens have been used[2] but the majority of observers[6] are in favor of small to moderate doses of x-rays in the treatment of acute inflammations. Heidenhain and Fried[7] advised 6 to 15 per cent of the erythema dose for inflammatory conditions in children. In a comprehensive review of radiotherapy for inflammatory processes Desjardins[5] concluded that small doses ranging between 10 and 50 per cent of the erythema dose repeated at four- to eight-day intervals gave the best results. He stressed the point "that treatment should not be confined to the area visibly or palpably affected but should extend well beyond such limits and include an adjacent zone of normal territory." We have employed 10 to 20 per cent of the

TABLE I

HOSPITAL CASES TREATED WITH ROENTGEN RAYS

NAME	SEX	AGE (YR.)	ASSOCIATED CONDITIONS	DURATION OF ADENITIS BEFORE THERAPY (DAYS)	NUMBER OF TREATMENTS	TOTAL DOSE OF ALL FIELDS (R)	FEVER POST-THERAPY (DAYS)	RESULTS
R. R.	M	8	Tonsillitis, nasopharyngitis, purulent otitis media: 103.3° F.	2	2	120	3	Resolution in 11 days.
J. A.	M	8	Upper respiratory infection: 102.6° F.	2	1	80	2	Resolution in 11 days.
D. W.	M	3	Tonsillitis, hemorrhagic nephritis: 102.9° F.	3	2	80	7	Resolution in 16 days.
N. G.	F	7	Tonsillitis, nasopharyngitis: 103.4° F.	3	1	80	3	Resolution in 9 days.
J. D.	M	4	Tooth extraction, cellulitis of neck: 103.6° F.	6	2	120	1	Resolution in 13 days.
R. T.	M	3½	Impetigo of scalp: 103.7° F.	6	3	240	5	Resolution in 20 days, impetigo difficult to eradicate.
W. B.*	M	2	Peritonsillar abscess (?): 103.8° F.	7	5	300	†	Resistant: unaffected.
B. D.	M	11	Tonsillitis: nasopharyngitis: catarrhal otitis media: 101.8° F.	10	3	180	3	Suppuration in 5 days.
R. R.	F	11	Tonsillitis: 103° F.	11	3	180	12	Resolution in 15 days.
A. P.	F	13	Tonsillitis: 102.9° F.	12	2	140	7	Resolution in 12 days.
E. N.	M	2	Tonsillitis: nasopharyngitis: acute nephritis: 104.2° F.	21	1	60	5	Suppuration 4th day: hemolytic streptococcus.
J. N.	F	6	Upper respiratory infection: 101.5° F.	2	2	184	3	Suppuration 3rd day: hemolytic streptococcus.
J. J.	M	6	Tonsillitis: 102° F.	5	2	150	4	Resolution in 10 days.
R. G.	M	4	Acute pharyngitis: 100.8° F.	7	1	80	1	Resolution in 4 days.
K. G.	M	3	Suppurative otitis media: 102° F.	21	2	221	3	Resolution in 23 days.
H. R.	M	2½	Acute pharyngitis: 103° F.	6	1	80	2	Resolution in 2 days.
T. B.	M	1½	Suppurative otitis media, aphthous stomatitis: 101° F.	28	4	296	1	Resolution in 10 days.
H. B.	M	8	Upper respiratory infection: 101.5° F.	14	3	216	1	Resolution in 3 days.
J. Z.	M	4	Upper respiratory infection: 102° F.	2	3	267	2	Resolution in 8 days.
M. J.	F	11	Tonsillitis, nasopharyngitis: 101° F.	5	3	150	2	Resolution in 12 days.
R. McG.	M	3	Suppurative otitis media; upper respiratory infection: 102° F.	2	3	150	2	Resolution in 12 days.

* See discussion.

†Continued low grade fever.

TABLE II

HOSPITAL CASES TREATED WITH MEDICAL MEASURES

NAME	SEX	AGE (YR.)	ASSOCIATED CONDITIONS	DURATION BEFORE THERAPY (DAYS)	FEVER AFTER THERAPY (DAYS)	RESULTS
E. P.	M	2	Upper respiratory infection: 103° F.	1	8	Still had enlargement when taken home on 8th day.
B. J.	M	8	Tonsillitis, nasopharyngitis: 101.8° F.	3	13	Suppuration 11th day: hemolytic streptococcus.
W. H.	M	5	Gingivitis, nasopharyngitis, acute nephritis: 102.9° F.	3	10	Suppuration 9th day: hemolytic streptococcus in blood stream and gland. Complete recovery.
C. W.	M	8	Influenza (?), nonsuppurative arthritis: 103.8° F.	4	9	Resolution in 36 days.
E. T.	F	3	Tonsillitis: 102.4° F.	5	6	Suppuration 6th day: *Staphylococcus aureus*, nonhemolytic streptococcus.
B. C.	F	6	Tonsillitis: 102° F.	6	18	Resolution in 19 days: hemolytic streptococcus in throat.
D. R.	F	6	Tonsillitis, headache, neck stiffness: 104.6° F.	10	19	Resolution in 32 days.
C. S.	F	2¾	Impetigo of scalp, purulent otitis media: 103.3° F.	14	9	Resolution in 12 days.
T. H.	M	8	Tonsillitis, nasopharyngitis: 102.5° F.	14	3	Suppuration on 8th day: *Staphylococcus aureus.*
S. E.	F	3¾	Tonsillitis, acute nephritis: 103.6° F.	21	6	Resolution in 15 days.
B. M.	M	8	Acute tonsillitis, Vincent's angina: 100.5° F.	2	3	Resolution in 7 days.
B. H.	F	7	Pharyngitis: 100° F.	3	2	Resolution in 4 days.
M. V.	M	8	Tonsillitis, right otitis media: 100° F.	3	10	Resolution in 13 days.
A. G.	F	6	Upper respiratory infection: 102° F.	2	*	Suppuration on 4th day: *Staphylococcus aureus.*
R. C.	M	7	Upper respiratory infection: 102° F.	10	3	Suppuration on 12th day: hemolytic streptococcus.
D. M.	F	1	Upper respiratory infection, pyuria: 101° F.	12	15	Suppuration on 7th day: *Staphylococcus aureus.*
E. K.	M	7	Pharyngitis, conjunctivitis: 104° F.	4	18	Resolution in 22 days.

*Continued low grade fever.

TABLE III

COMPARISON OF HOSPITALIZED CASES TREATED WITH ROENTGEN RAYS AND OTHER MEASURES

	ROENTGEN THERAPY	OTHER MEASURES
Number of cases	21	17
Sex (males)	16	9
Age (years)	1½ to 13 (Average = 5.8)	2 to 8 (Average = 5.6)
Initial temperature (° F.)	100.8-104.2 (Average = 102.5)	100-104.6 (Average = 102.3)
Duration before therapy (days)	2 to 28 (Average = 8.3)	1 to 21 (Average = 6.9)
Fever postherapy* (days)	1 to 12 (Average = 3.6)	2 to 19 (Average = 9.5)
Result:		
Suppuration	3 (14.3%)	7 (41.2%)
Resolution	17 (80.9%)†	10 (58.8%)

*One patient in each series continued to run a low grade fever until discharge from the hospital.

†One case was uninfluenced by radiation therapy.

erythema dose repeated every three to five days and the field exposed has always included normal tissues surrounding the involved lymph nodes.

One of the most striking results of roentgen therapy was the rapid improvement in the constitutional symptoms. A pronounced drop in temperature occurred in twelve to forty-eight hours with marked relief of pain and discomfort in the majority of cases. Irritability was diminished, appetite improved, and better rest was promoted. The swelling receded and resolution was complete in a shorter time than occurred when the usual medical procedures were employed. In an occasional instance, within a few hours after radiation, there was a temporary exacerbation of symptoms with increased temperature and local swelling. This reaction subsided within twelve to twenty-four hours and no untoward effects remained. With the small dose of x-rays employed symptoms of radiation sickness, such as nausea and vomiting, were not encountered. Skin reactions did not occur.

Resolution occurred in 17 (80.9 per cent) of the roentgen treated cases as opposed to 10 (58.8 per cent) in the group for which other measures were employed. Suppuration resulted in 3 (14.3 per cent) of the roentgen treated cases, while 7 (41.2 per cent) of the control group showed abscess formation. One case was uninfluenced by five roentgen treatments. This patient, a white male two years of age, was thought to have a peritonsillar abscess. However, incision yielded only blood. He continued to run a low grade fever and the glandular swelling persisted despite irradiation. Biopsy of a lymph node showed chronic inflammatory changes with considerable leucocytic infiltration. Thus the lack of response was not due to an absence of leucocytes, a factor held responsible by some authors for the

failure of the roentgen rays in occasional cases of acute lymphadenitis. Previously Desjardins and others have noted instances where roentgen therapy has not proved beneficial. However, there is universal agreement[5, 8] that no unfavorable or detrimental effect results.

Table IV summarizes the results of roentgen therapy in 41 cases of acute cervical adenitis treated in the out-patient clinic. In this group ranging in age from seven months to fourteen years a dose of 70 to 200 r was administered at two- to seven-day intervals. One to three treatments were usually sufficient to bring about resolution which occurred in 35 (85.4 per cent) cases. Suppuration occurred in 6 (14.6 per cent) instances. In 5 of these patients radiation was not begun until relatively late in the course of the disease (2 in the second and 3 in the third week or later). One case was semifluctuant when first seen and x-rays were used to hasten localization.

Our findings concerning the value of x-rays in the treatment of acute cervical adenitis are in accord with those of other authors. Heidenhain and Fried[7] reported 15 cases with good results in 14; only one was uninfluenced. In Rosenberg's series of 80 children, all under seven years of age, a dose of 260 r was employed once weekly and resolution occurred in 85 per cent. Cures were reported in 85.7 per cent of 105 cases, ranging in age from six months to eleven years, treated by Schenck.[3] He employed 25 to 50 per cent of an erythema dose repeated at four- to five-day intervals, giving a maximum of three treatments. More than 85 per cent of the cases in the combined series of 355, referred to by Schenck, terminated in resolution. One is impressed by the uniformly favorable results reported by various observers and it is our belief that roentgen therapy should be administered to all cases of acute cervical adenitis in which there is any suspicion of a suppurative termination, or where other measures have previously failed to cause improvement.

The extreme sensitiveness of lymphoid cells to the x-rays makes acute lymphadenitis the most responsive of all inflammatory conditions. The exact mode of action of roentgen rays on inflamed tissues has not been conclusively established. However, it is believed by some[9] that radiation in small or moderate doses is stimulative and augments the inflammatory process. Cellular infiltration and phagocytosis are increased and leucocytic destruction with the liberation of protective substances is promoted. All stages of the inflammatory process are shortened and termination is accelerated. Roentgen therapy is most beneficial in the early stages of inflammation before necrotic foci develop. Here retrogression is rapid and absorption is the final result. If treatment has been delayed until suppuration has already begun it will accelerate the necrosis, cause rapid localization, and prevent spread to neighboring glands.

TABLE IV

OUT-PATIENTS TREATED WITH ROENTGEN RAYS

CASE	SEX	AGE (YR.)	ASSOCIATED CONDITIONS	DURATION OF ADENITIS BEFORE THERAPY (DAYS)	NUMBER OF TREATMENTS	TOTAL DOSE OF ALL FIELDS (R)	RESULTS
1	M	3	Chronic tonsillitis	42	4	600	Resolution.
2	F	3	Upper respiratory infection	6	3	273	Resolution in 16 days.
3	M	3	Tonsillitis, otitis media, nephrosis	2	3	300	Resolution in 7 days.
4	F	14	Abscessed tooth	5	2	160	Resolution.
5	F	13 mo.	Tonsillitis	10	1	73	Suppuration in 6 days.
6	F	5	Tonsillitis	7	5	375	Resolution.
7	F	8 mo.	Upper respiratory infection, otitis media	1	2	150	Resolution in 13 days.
8	F	2½	Tonsillitis	15	2	142	Resolution in 10 days.
9	M	7 mo.	Upper respiratory infection	4	1	75	Resolution.
10	M	14 mo.	Tonsillitis	7	1	75	Resolution in 6 days.
11	M	15 mo.	Upper respiratory infection, otitis media	7	2	146	Resolution in 8 days.
12	M	3½	Tonsillitis	7	5	375	Resolution in 30 days.
13	M	3½	Upper respiratory infection	9	1	150	Medical measures failed previously. Resolution in 12 days.
14	M	14	Abscessed tooth	30	4	304	Resolution.
15	F	9	Hordeolum	5	2	144	*Staphylococcus aureus.* Suppuration in 4 days (fluctuant when first treated).
16	F	2	Post mumps (?)	21	3	380	Resolution in 15 days. Resisted previous medical treatment.
17	F	5	Axillary adenitis	4	4	600	Resolution in 14 days.
18	M	1½	Upper respiratory infection	3	1	72	Resolution in 9 days.
19	M	3	Tonsillitis, nasopharyngitis	13	1	150	Resolution.
20	M	3	Stomatitis	14	3	384	Resolution in 14 days; did not respond to previous medical treatment.
21	F	2½	Tonsillitis	8	3	280	Resolution in 11 days.

TABLE IV—CONT'D

CASE	SEX	AGE (YR.)	ASSOCIATED CONDITIONS	DURATION OF ADENITIS BEFORE THERAPY (DAYS)	NUMBER OF TREATMENTS	TOTAL DOSE OF ALL FIELDS (R)	RESULTS
22	F	3	Upper respiratory infection	1	2	148	Resolution.
23	F	7	Tonsillitis	1	1	100	Resolution in 4 days.
24	M	5	Upper respiratory infection	5	5	390	Resolution in 91 days.
25	M	6	Tonsillitis	21	1	100	Suppuration in 2 days.
26	M	4	Tonsillitis, nasopharyngitis	7	3	216	Resolution in 13 days.
27	M	2⅔	Streptococcus sore throat	30	2	142	Suppuration in 3 days.
28	M	2	Pharyngitis	13	2	144	Resolution in 8 days.
29	F	13 mo.	Tonsillitis	7	2	142	Resolution in 14 days.
30	M	12	Tonsillitis	7	3	300	Resolution in 16 days.
31	F	2	Tonsillitis	1	1	150	Resolution.
32	F	16½	Cellulitis, tonsillitis	30	2	350	Suppuration in 12 days; culture negative.
33	M	8	Tonsillitis, glomerulonephritis, dental caries	4	2	300	Resolution in 12 days.
34	M	2½	Otitis media, upper respiratory infection	3	5	470	Resolution in 16 days.
35	M	5 mo.	Tracheobronchitis	2	3	300	Resolution in 10 days.
36	M	4	Upper respiratory infection	6	2	300	Resolution in 14 days.
37	M	7	Post measles	14	2	280	Resolution.
38	F	4½	Upper respiratory infection	10	3	350	Suppuration in 9 days. *Staphylococcus albus.*
39	F	5½	Tonsillitis	10	3	287	Resolution in 15 days.
40	F	18 mo.	Impetigo	5	2	172	Resolution.
41	M	7	Post measles	14	2	280	Resolution.

SUMMARY

Sixty-two children with acute cervical adenitis were treated with small doses of roentgen rays. Resolution occurred in 52 (83.9 per cent) cases, while 9 (14.5 per cent) terminated in suppuration. In a group of 21 adequately controlled hospitalized patients who were irradiated, 17 (81 per cent) were cured as opposed to 10 (58.8 per cent) treated by other measures. Suppuration resulted in 3 (14.3 per cent) of the cases treated with x-rays as opposed to 7 (41.2 per cent) in the control group. Rapid subjective improvement and accelerated termination are striking features of roentgen therapy. Radiation is most beneficial in the early stage of inflammation where absorption is the usual final result. If suppuration is already present before treatment is started, necrosis is hastened and early centralization of the infection results. Surgical intervention is minimized and the cosmetic effect greatly enhanced.

CONCLUSION

Roentgen therapy is a most valuable aid to the treatment of acute cervical adenitis in children. It is the method of choice in all patients with marked glandular swelling.

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LYMPHATIC LEUCEMIA WITH PERTUSSIS

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IN 1936 Morris and Hurwitz[1] reported two cases of myelogenous leucemia associated with pertussis. These occurred in male infants of twenty-seven and nine months, respectively. The older patient had a generalized adenopathy, splenomegaly, and hepatomegaly. He contracted pertussis which was followed by a myelogenous leucemia with a white blood count of 150,000. The second case had pertussis at seven months. After a stormy convalescence of several weeks, splenohepatomegaly with a white blood count of 220,000 was observed. They stated that: "these are the only reports in the literature, of which the influence of whooping cough seems to have a direct relationship to the leucemia which followed."

It is significant that lymphatic leucemia does not appear with whooping cough even though the literature appears to be replete with reports of whooping cough associated with various other diseases. Tuberculosis, syphilis, malaria, the various exanthems, and other infectious diseases are quite frequently seen with pertussis.[2] We thought, therefore, that because of its rarity this case was worthy of being reported.

CASE REPORT

C. G., a seven-year-old white schoolboy, was admitted to the Morrisania City Hospital on July 14, 1935, because of severe pallor, rhinitis, sore throat, and cough. The pallor was noted by his mother two weeks prior to admission and was getting progressively worse. The cough, which was of ten days' duration, was productive of a mucoid material. His general health had been more or less poor for several months prior to the present illness, as manifested by anorexia, no gain in weight, and general asthenia. The past history was normal except for measles, chickenpox, and frequent colds. The family history revealed no evidence of any chronic diseases.

Physical examination on admission disclosed a child who seemed to be suffering from some chronic illness. He was markedly underweight, and his pallor was extreme. Over the right sacroiliac region and the left lower extremity were ecchymotic spots. The cervical, axillary, and inguinal glands were enlarged. The eyes reacted normally to light and accommodation. The conjunctivae were pale, but no petechiae were found. The buccal mucous membrane was pale, the tongue coated, and the tonsils were enlarged and cryptic. No areas of ulceration were found in the oral cavity or pharynx. The apex beat of the heart was at the nipple line in the fifth interspace, and no adventitious sounds were heard, nor were there any irregularities present. Diminished percussion resonance was observed over the right upper pulmonary field and over a patch posteriorly adjacent to the left scapula. Bronchovesicular to bronchial breathing with fine crepitant râles was in evidence over the area of dull-

From the Pediatric Service of Dr. Louis H. Barenberg, Morrisania City Hospital.

TABLE I

SUMMARY OF THE BLOOD FINDINGS

DATE 1935	PER CENT SAHLI HG	R.B.C. M.	W.B.C.	POLY-MORPH. NEUT.	LYMPHO-CYTES	EOSIN-OPHILES	BASO-PHILES	MONONU-CLEAR	ABNORMAL CELLS	PLATE-LETS	CLOTTING TIME: MINUTES	BLEEDING TIME: MINUTES
7/14	31	2.01	230,400	3	97	0	0	0	0	90,000	4.0	35.0
7/18	47	2.23	88,000	6	94	0	0	0	0	110,000	4.5	30.0
7/23	49	2.42	41,250	8	92	0	0	0	0	155,000	4.0	14.5
7/27	57	2.85	32,500	21	79	0	0	0	0	192,000	4.0	7.5
7/31	62	3.76	19,350	24	75	0	0	1	0	330,000	4.0	3.5
8/ 5	77	4.29	10,550	36	62	1	0	1	0	285,000	4.0	4.5
8/ 9	68	4.54	11,000	50	48	0	1	1	0	217,000	4.0	5.0
8/14	78	3.84	7,950	38	62	0	0	0	0	280,000	4.5	4.0
8/22	71	3.25	7,600	68	32	0		0	0	260,000	6.0	4.5
8/29	84	4.55	7,900	42	53	3	1	1	0	190,000	2.0	3.5
9/28	98	3.69	68,000	3	93				4 lymphoblasts			
11/14	32	1.71	1,550,000	1	65	0	0	2	32 lymphoblasts			
11/15	22	1.22	90,000	2	77				21 lymphoblasts	90,000	8.0	2.5

ness. Abdominal examination revealed a hugely enlarged liver. Its edge was felt just below the level of the umbilicus and was somewhat tender. The spleen was enlarged down to 2 cm. below the left costal border. The remaining examination did not yield any unusual features. The routine blood count done on admission presented a marked anemia (hemoglobin 31 per cent, red blood count 2.01 m.) and an extreme leucocytosis of 230,400 white cells, of which 97 per cent were lymphocytes. This clinical picture was noted on the eleventh day of illness.

The enormous white cell reaction and marked anemia suggested very strongly the possibility of an acute blood dyscrasia, particularly because of the presence of an enlarged liver, spleen, and lymph glands. However, on the next day, the cough, which was previously hacking in character, seemed to be somewhat spasmodic and paroxysmal. This feature of the syndrome stimulated the thought of pertussis being the etiologic factor in producing the hemogram. Cough plates were taken and sent to the Bureau of Laboratories of the Department of Health, New York City, for culture and examination. They were reported as positive for *B. pertussis.* Laboratory data were all negative except for the blood picture. Radiographic studies of the chest, skull, and long bones were all normal.

On July 15, or the second day after admission, he was given a transfusion of 300 c.c. of citrated blood, by the gravity method. This was soon followed by a chill and rise of temperature up to 105.2° F. This reaction was short lived, and in twenty-four hours improvement in his general condition was evident. During his hospitalization he went through a typical and severe course of whooping cough. As the condition improved, his pallor became less marked, the liver and spleen receded, and he appeared to be alert. Following the transfusion the boy made a slow but gradual recovery. He was discharged on Aug. 14, 1935, markedly improved. At this time he was coughing only slightly, his blood picture was practically normal, and he had gained several pounds in weight. He weighed 39¼ pounds on admission and 46 pounds at the time of his discharge. The diagnosis subsequently was whooping cough and extreme hyperleucocytosis. This was entertained because of the type and characteristic features of the cough, the clinical course of the disease during hospitalization, the positive cough plates, the positive white cell curve, and the negative hemogram typical of acute leucemia. However, this diagnosis was made with reservations because of the possibility of the case being a masked leucemia. The procedure, therefore, was to follow the blood reactions after discharge from the hospital.

The patient returned for follow-up examinations at weekly and later at biweekly intervals (Table I). The blood count continued to be normal until September 28, when the white cell count rose to 68,800 with a differential count of 93 per cent lymphocytes, 4 per cent lymphoblasts, and 3 per cent polynuclear neutrophiles. This was the first indication hematologically of a blood dyscrasia. He disappeared after this examination and efforts to find him were futile.

The child was readmitted to the pediatric ward on Nov. 14, 1935, with an interval history of progressive asthenia, abdominal pain in the left upper quadrant, and bleeding from the mouth. Physical examination at this time showed an acutely ill child with pronounced anemia. Slight exophthalmos and pallor of the conjunctival mucous membranes were apparent. Ophthalmoscopic examination revealed a bilateral papilledema and venous engorgement. The teeth were carious, and the tongue was coated. No ulcerations or leucemic infiltrations were seen in the mouth. A raised erythematous area with a central necrotic patch was noted on the chin. Small bilateral cervical glands were present. The liver was firm, not tender, and palpable three fingerbreadths below the costal margin. The spleen was very tender, moderately firm, notched, and felt at the level of the umbilicus. Bony tenderness was

elicited over the right lateral malleolus and tibia. A shower of purpuric spots could be seen over this area. A transfusion of 275 c.c. of citrated blood was given on November 15 by the gravity method. The child became increasingly irritable, pale, and drowsy. He had repeated epistaxis and facial twitchings. Hematemesis and melena folowed, and he lapsed into coma and died on Nov. 16, 1935.

NECROPSY

Examination was performed one hour after death.

Gross Examination.—The body was that of a poorly nourished, anemic looking, white, male child. Numerous small purpuric hemorrhages were scattered over the chest, back, abdomen, and lower extremities. There was dried blood over the teeth, gums, left nostril, and ears. Localized hemorrhages were noted in the various layers of the scalp. The meninges and brain were pale and edematous but showed no evidence of purpura or hemorrhage. The cervical, submaxillary, supraclavicular, and thymus glands were moderately enlarged, soft, and pinkish red in color. The lungs were exceptionally pale, crepitant, and covered with numerous purpuric areas which extended into the parenchyma. The trachea and bronchi were clear. The hilus lymph nodes were markedly enlarged and matted together. The surface of the heart and endocardium disclosed numerous large petechial hemorrhages. The myocardium was pale and light brown. The valves were normal. The peritoneum and bowel were covered with small petechial hemorrhages. The liver was enlarged down to 5 cm. below the costal margin. It was firm and yellowish brown with numerous pinpoint hemorrhages over the surface. The gallbladder and bile ducts appeared normal. The spleen was slightly enlarged, soft, pinkish red, and had two small hemorrhages extending into the parenchyma from the surface. The malpighian bodies were increased in size and number and appeared to be scattered profusely through the pulp. The kidneys were pale and had a few pea-sized infarcts on the surface. These had whitish centers with hemorrhagic peripheries. The cortex and medulla were normal in size and there were no gross infiltrations of leucemic tissue. The ureters, bladder, and prostate showed no hemorrhagic areas. The testes were soft and pinkish red. The gastrointestinal tract contained a large amount of bloody material and was markedly distended. Peyers patches were greatly enlarged, granular, and reddish and appeared to be especially prominent in the ileocecal region. No ulceration of the mucosal surface was seen. The mesenteric and retroperitoneal lymph nodes were enlarged, soft, and pinkish red. The bone marrow of the sternum, ribs, and vertebrae was jellylike and light brown.

Microscopic Examination.—Kidney: The capsule of the kidney was very thin and was not infiltrated with lymphocytes. A small subcapsular hemorrhage was noted with a surrounding region containing many lymphocytes. There were numerous collections of lymphocytes in the interstitial tissue surrounding the blood vessels, tubules, and glomeruli. A few scattered hemorrhages were surrounded by solid collections of lymphocytes. The cells of the convoluted tubules showed marked cloudy swelling. The glomeruli were normal except for an increase of lymphocytes in the tufts (Fig. 1).

Spleen: The capsule of the spleen was not infiltrated with lymphocytes. The malpighian bodies were greatly increased in size and contained large lymphocytes in their central portions while the peripheral zone contained innumerable small lymphocytes closely packed together. The pulp sinuses and venules were filled with many large and small lymphocytes which replaced and crowded out the normal splenic cells. Lymphocytes were scattered profusely throughout the pulp. There were a few small hemorrhages surrounded by lymphocytes (Fig. 2).

Fig. 1.—Kidney: Shows the numerous collections of lymphocytes in the interstitial tissue and cloudy swelling of the convoluted tubules.

Fig. 2.—Spleen: Shows innumerable small lymphocytes replacing pulp sinuses.

Fig. 3.—Heart: Shows the collections of small lymphocytes in the interstitial tissue between the muscle fibers; in areas partial fragmentation may be seen.

Fig. 4.—Liver: Shows numerous collections of lymphocytes in the periportal areas encroaching on the portal vein, hepatic artery and bile ducts.

Heart: There were numerous collections of small lymphocytes in the interstitial tissues between the muscle fibers. Some of these were in the capillaries and others were loose in the connective tissue. Many of these collections were large and surrounded by small hemorrhages of fresh red blood cells which crowded the adjacent muscle fibers to one side and even caused partial fragmentation. No degenerative changes were seen in the muscle fibers (Fig. 3).

Liver: The capsule was infiltrated with small lymphocytes. Numerous collections of lymphocytes were observed in the periportal areas which encroached on the portal vein, hepatic artery, and bile ducts but did not involve their walls. The sinusoids contained a few scattered lymphocytes. Cloudy swelling was evident in the liver cells (Fig. 4).

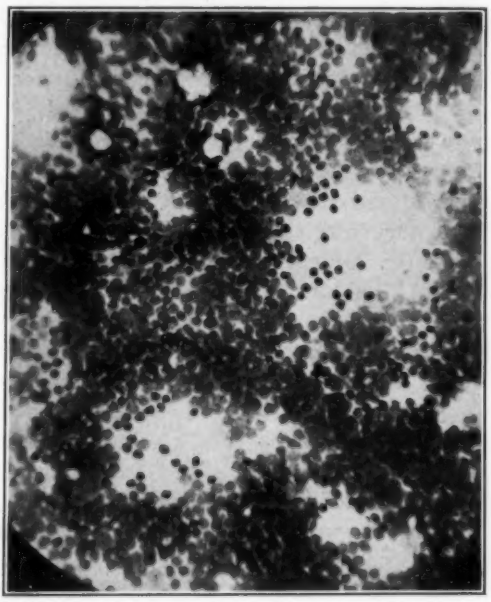

Fig. 5.—Bone Marrow: Shows the replacement of the normal marrow elements by lymphocytes and lymphoblasts.

Bone Marrow: The marrow elements, such as red blood cells, granular leucocytes, and megakaryocytes were replaced by many small and large lymphocytes and lymphoblasts (Fig. 5).

Thymus: The entire organ was completely filled with small and large lymphocytes. These obscured the normal thymic tissue and cells.

Lymph Nodes: The entire architecture of the glands was distorted by lymphatic infiltration and hyperplasia.

Adrenals: There was a marked increase and accumulation of lymphocytes in the medulla.

Testes: There was a marked infiltration of small and large lymphocytes in the interstitial tissue compressing the seminiferous tubules.

Ileum: There was marked lymphocytic infiltration in the mucosa, submucosa, and muscularis.

COMMENT

Clinically, the question of the early diagnosis of pertussis is more or less common knowledge and is well summarized by Evans,[3] who is of the opinion that practically pathognomonic of pertussis are the absence of a previous history, an obstinate common cold, a dry cough becoming paroxysmal and worse at night followed by vomiting, the history of contact, the absence of an obvious cause in the throat or lungs, the presence of a frenal ulcer, and the absence of anorexia. However, without the usual confirming blood examination, all of these findings are very frequently misleading in the early stages. Daughtry-Denmark[4] found that complement fixation tests, using *B. pertussis* as the antigen, were positive in more than 90 per cent of cases of whooping cough even before the severe cough developed.

The diagnostic significance of hemograms in pertussis has been a subject of much controversy. It is common knowledge that pertussis may be responsible for a leucocytosis with a relative lymphocytosis, which may simulate the hemogram observed in lymphatic leucemia. When it is impossible to culture the Bordet-Gengou bacillus with the aid of a cough plate, such a typical blood picture may be of significance before the onset of the paroxysmal stage. Finkelstein[5] does not attach any importance to the blood count in the catarrhal stage. His observations would indicate that an increased lymphocyte response should not be expected until the paroxysms appear. Schmal and his collaborators[6] saw characteristic blood findings in many instances in the catarrhal stage. They also found normal hemograms in cases which subsequently developed pertussis. Dolgopol[7] described an interesting phenomenon pertaining to the lymphocyte curve in pertussis. She observed a marked increase in the total number of lymphocytes which was most apparent at the height of the paroxysmal stage. There was no increased lymphocyte response during the catarrhal stage.

The differentiating features between the lymphocyte of whooping cough and acute leucemia rest in its size and maturity. In pertussis they are usually small and show no evidence of immaturity, while in acute lymphatic leucemia just the reverse is true. However, in cases of chronic leucemia during a remission when the white count is only fairly high, ranging between forty and sixty thousand or less, the lymphocyte may have normal morphologic and staining characteristics. In these cases diagnosis is extremely difficult if not impossible. Blood studies must, therefore, be made at regular intervals until such evidence appears as to make the diagnosis tenable. In other words it is evident from the previous discussion that the differentiating features do not depend so much on the number of lymphocytes as on the size and maturity of the cells.

At first we assumed that our case was one of extreme hyperleucocytosis sometimes encountered in whooping cough. An explanation for such a blood picture was sought. Thelander, Henderson, and Kilgariff[8] reviewed the literature of fourteen cases of hyperleucocytosis in pertussis and added five additional cases (Table II). In seeking an explanation for this phenomenon in pertussis, varying opinions have been advanced. Meunier[9] disproved the theory of mechanical extrusion of cells caused by the paroxysms, by finding no variation in the number of leucocytes

TABLE II

SUMMARY OF THE CASES OF PERTUSSIS WITH HYPERLEUCOCYTOSIS REPORTED IN THE LITERATURE

AUTHOR	REFERENCES	NUMBER OF CASES	AGE OF PATIENT	EXTREME LEUCOCYTOSIS	% LYMPHOCYTES	ABNORMAL CELLS
Cabot, R. C.	Clin. Exam. of Blood, N. Y., Wm. Wood & Co., 1904	1	15 mo.	185,000	64.5	None
Stevens, J. L.	Brit. M. J. 2: 791, 1902	1	22 mo.	227,810	50.2	None
Crombie, J. F.	Edinburgh M. J. 1: 222, 1908	2	20 mo.	233,000	67.0	None
			3 yr.	120,000		None
Reiche, F.	München. med. Wchnschr. 67: 1352, 1920	1		172,000	43.6	None
Churchill, F. S.	J. A. M. A. 46: 1506, 1906	1	4 yr.	112,000		None
Austrian, C. R.	Bull. Johns Hopkins Hosp. 22: 296, 1911	1	4 yr.	206,000	24.0	None
Bourne, G., and Scott, J. M.	Brit. M. J. 1: 386, 1922	1	21 mo.	176,000	66.0	None
Strauss, cited by Simon, C. E.	Clin. Diag., Lea and Febiger	1		236,000	65.0	None
Seitz, R. P.	Am. J. Dis. Child 30: 670, 1925	2	8 mo.	192,000		None
				162,000		None
Nicholson, W. W.	Kentucky M. J. 30: 92, 1932	1		198,000	89.0	None
Thelander et al.	J. PEDIAT. 2: 288, 1933	5				None
Mitchell, A. G., and Friedman, L.	Arch. Pediat. 43: 617, 1926	1	4 mo.	233,600	48.0	None
Cooke, E. P.	Arch. Pediat. 47: 334, 1930	2	11½ mo.	158,400	89.0	None
			1 yr.	142,600	60.0	None
Frank, M.	Monatschr. f. Kinderh. 43: 420, 1924			132,000		None
Abt, A. F.	M. Clin. North America 14: 1229, 1931			213,300		None

in the peripheral blood before and after the whoop. Mishulow[10] confirmed the work of Bordet showing that the pertussis bacillus produces a toxin which may cause the stimulation of the leucocyte count. The lipoid fraction of the bacillus has also been held responsible for the leucocytosis with a lymphocytosis. It is questionable whether this reaction is specific for the pertussis organism. In view of the fact that *B. pertussis* might be found in the cilia of the respiratory tract, the amount of toxin absorbed might be in direct proportion to the number of bacilli found at the various levels of the tract. Above the larynx, where

there are very few organisms, little absorption occurs, whereas low down, as in a case cited by Mallory and Horner,[11] the bronchioles were covered with large numbers of pertussis bacilli. Absorption of excessive amounts of toxin from this site into the blood stream might offer an explanation for the extreme hyperleucocytosis.

It was only by repeated blood studies that we established the fact that our case was one of lymphatic leucemia. Unfortunately we were not familiar with the blood picture before admission. Clinically, however, the evidence in favor of a potential leucemia was manifest by the history of progressive pallor before the onset of the cough, the marked secondary anemia, and the hepatosplenomegaly associated with a generalized glandular enlargement. However, hyperplasia of the reticuloendothelial system is frequently seen with a resulting hyperleucocytosis. Abnormal cells are not observed in these syndromes. Consequently we are not justified in considering these cases in any other light than that of a potential leucemia. These patients improve spontaneously, as did our case. Unfortunately, because of the inherent instability of the reticuloendothelial system, they subsequently developed typical acute leucemias.

Following whooping cough, children usually show a marked improvement in their physical condition which is seen concomitantly with the return of the blood count to its normal level. It is interesting to note that our child showed a similar response during his convalescence.

It is unusual for a child to have a chronic lymphatic leucemia with remissions such as are seen in adults. We are not prepared to state that our case was one of chronic leucemia with a remission. If abnormal lymphocytes had been found before his first admission to the hospital or during his whooping cough, we would be justified in reporting a case of chronic lymphatic leucemia with remissions. However, it must be obvious that many arguments might be advanced in favor of considering this report as an example of acute lymphatic leucemia in a child who had exhibited an unstable reticuloendothelial system. Whether or not the whooping cough was responsible for the acute lymphatic leucemia which followed, we are not able to state. Our experience in this case emphasizes the importance of repeating the hemograms in any case of pertussis with hyperleucocytosis. It is only by such a careful follow-up procedure that a correct diagnosis of leucemia can be ascertained. Bone marrow biopsy is a valuable aid in making an early diagnosis in obscure cases. Unfortunately permission was not granted to perform this procedure in our case. The reduction in the number of platelets is a constant finding in lymphoid leucemia. It is worthy to note that only on two occasions did we find platelet counts which were below normal standards. In the first instance no abnormal white cells were found associated with this marked reduction in platelets (Table I). It is reasonable to assume that the reported instances of pertussis with hyper-

leucocytosis may have been potentially leucemic in nature. Repeated blood studies on these patients, following their pertussis, might have revealed several instances of blood dyscrasias.

SUMMARY

A case of lymphatic leucemia with pertussis is presented, and the absence of case reports of lymphatic leucemia with pertussis is emphasized. The difficulties encountered in differentiating lymphocytic reactions in acute lymphatic leucemia and pertussis are discussed. Attention is called to the confusion that may arise in differentiating between a remission of a lymphatic leucemia and a case of convalescent whooping cough. Hyperleucocytosis in pertussis should be followed by repeated blood counts so that latent leucemias may be detected.

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THE INCIDENCE OF BREAST FEEDING IN A SUBURBAN METROPOLITAN AREA

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BREAST milk as the ideal infant food needs no defense. All physicians, be they specialists or general practitioners, bow before this living vital fluid and cannot accept with complacence the fact that more and more they must substitute artificial preparations. Grulee and his associates[1] have shown mortality and morbidity rates in artificially fed infants to be far above those of breast-fed infants. Others have pointed out that the frequent necessity of feeding infants food other than breast milk is not always accompanied by failure—that success, in fact, is the rule. Faber and Sutton[2] found breast feeding superior during the first three months, but their records show that after this period artificially fed infants lead in gain. They were unable to demonstrate evidence of increased resistance to infection in breast-fed infants, and stated that weaning after the third month was not in itself to be feared. Glazier[3] pointed out that infant mortality and morbidity are definitely related to economic and hygienic conditions and that in districts where these conditions were good the average bottle-fed baby does as well as the breast-fed. Garland and Rich[4] stated that the social status, the intelligence, and the environment of the mother are more important factors in assuring the health and well-being of the child than is the type of feeding. Lamb[5] says, "It is quite proper to encourage breast feedings, but we should not make the mistake of disparaging substitute feedings, for . . . the babies fed on artificial milk formulas have shown no serious deviation from that normal development that regularly follows adequate breast feedings." This states my own feeling in the matter.

However, the impression all physicians give in writing of the success of artificial feedings is that of being on the defensive. Facing the fact that the preferred breast feeding is not always possible, they concentrate on success without it, and their very success results in an attitude of indifference, too easily transmitted to the layman, toward the frequent inability of mothers to nurse. Hegar[6] attributes this inability to the influence of generations of undervaluing maternal feeding.

How frequent in a suburban metropolitan area in the last ten years is this inability to nurse? The answer to this question has been sought in the study of 437 cases, random selections from the case records of the author. All were followed as private cases from the neonatal period throughout infancy. The duration of nursing was determined from personal knowledge, and not from a mother's statement secured months or years after her experience with infant feeding. The accuracy

of investigations based on the latter type of information may well be questioned. All infants in the present series were born in a hospital, the great majority delivered by a physician limiting his practice to obstetrics and declining all responsibility for the baby. In this circumstance, it would seem, lies one of the factors in the decline of breast feeding: care of mother and babe is divided between two persons. Whose is the responsibility in such essential matters as care of the nipples, condition of the breasts, encouragement and instruction of the mother? Who shall insist on an honest trial of breast feeding?

The incidence of breast feeding in the 437 cases studied is best shown in Table I. The percentages make a poor showing. The mothers, most of them American, are persons of probably more than average intelligence and training, living in what may be considered adequate financial circumstances. The great majority are willing and anxious to nurse their babies, of this I feel sure. I do not believe we can fall back on the defense of lack of desire or cooperation. It is true, however, that mothers are often unwilling to continue mixed feedings, that they insist on "all or none," and for this I cannot much blame them. Mixed feedings are a hard task for all concerned, and I agree with Faber and Sutton[2] when they state that "if the baby cannot get at least half of his food supply from the maternal breast he should be weaned at once, and be spared, with his mother, the annoyance and uncertainty of 'allaitement mixte.'" Yet there are mothers who must nurse only partially, or not at all. Many breasts just do not make breast milk.

TABLE I

INCIDENCE OF BREAST FEEDING IN 437 MOTHERS

TIME IN MONTHS	COMPLETE BREAST FEEDING	PARTIAL BREAST FEEDING	WEANED
To 1 month	38% (168 cases)	38% (164 cases)	24% (105 cases)
3 or longer	19% (82 cases)	25% (111 cases)	56% (244 cases)
6 or longer	7% (30 cases)	10% (42 cases)	83% (365 cases)
9 or longer	1.5% (7 cases)	4.5% (20 cases)	94% (410 cases)

Factors of interest in studying breast feeding are age and parity of the mother. For purposes of comparison, a scoring system for nursing mothers has been devised. Ten points are awarded for each month of complete nursing, five points for each month of partial breast feeding. Thus a concrete figure is obtained: for example, a mother nursing her infant completely for ten months scores 100; a mother nursing her infant completely for five months and partially for two months scores 60; a mother nursing her infant only partially for four months scores 20. Use of such a scoring system in other studies would facilitate comparisons.

TABLE II

SCORING SYSTEM FOR NURSING MOTHERS

Each month of complete nursing scores 10 points.
Each month of partial nursing scores 5 points.

A.

AGE	NUMBER OF CASES	AVERAGE SCORE
25 years or less	54	18.5
26 to 30	101	20.4
31 to 35	35	21.3
Over 35	9	26.0*

B.

Para i	255	20.3
Para ii	134	17.0
Para iii	34	15.7
Para iv	14	11.1
Total series	437	18.6

*Two scores of 80 and 90 distort this figure.

Age seems of little importance in the duration of nursing. It is interesting to note that of 199 mothers, 118 para i's averaged twenty-seven years of age. This is too old for first babies, but our social and economic conditions seem responsible. The ages of 66 para ii's averaged twenty-eight and one-half years; of 10 para iii's thirty and one-half years; and of 5 para iv's, thirty-three years. The scores for primiparas are superior to those of multiparas, but all are discouragingly low. The average score for the entire series is only 18.6.

Other important factors enter into the problem, crowded living conditions, loss of sleep, fatigue, sore nipples. In this connection attention is directed to the excellent study of English mothers by Pooler.[7] It seems worth while to present his table (Table III).

TABLE III

PRIMARY CAUSES OF FAILURE OF BREAST FEEDING
(FROM POOLER)

Domestic duties	33	
Domestic duties with depressed nipples	11	
Domestic duties with somnolence	1	
Domestic duties with too frequent feeds	12	
Domestic duties and mother badly fed	2	
Domestic duties and mother ill	2	
Domestic duties and weakling	1	
Domestic duties and depressed nipples	1	63
Nipple trouble, sore	4	
Nipple trouble, depressed	6	10
Exudative diathesis (dyspepsia)		3
Mother badly fed		1
Abscess of breast		2
Injury to child at birth		1
Weakling	7	
Weakling and depressed nipples	2	9
Cleft palate		2
Bad management during lying-in		9
		100

TABLE IV

AUTHOR	YEAR OF REPORT	NO. OF CASES	NOT NURSED (%)	TO 1 MO. (%)	3+ MO. (%)	6+ MO. (%)	9+ MO. (%)	REMARKS
Sedgwick[8]	1912	469	0.4	99.6	82	73	63	Wives of Minnesota physicians, by questionnaire
Koplik[9]	1912	1000	28				12	Consultation practice, from records
Mitchell[10]	1916	2800	20	80	55	42	34	Case records, Children's Hospital, Philadelphia
Brown[11]	1917	633	24	1 wk.+ 76	47	30	10	Private practice
Manning[12]	1920	1000	8	92	64	41	27	From private case records. 70% not seen until 9 mo.
Huenekens[13]	1924	3392	2	1 wk.+ 98	93.6	83.4	78.7	Results of 1920 breast feeding campaign, Minneapolis Infant Welfare
Hackett[14]	1927	100			84	76		Breast feeding campaign
Lamb[5]	1931	1000	7	93	70	57	29	Private practice
Weigard[15]	1934	560		1 wk.+	74			University of Nebraska Newborn Clinic
Grulee[1]	1934	20061					91	Chicago Infant Welfare Society, 10% American, 90% foreign born
Author	1936	437	24	76	44	17	6	Private cases

He felt that the return to domestic duties was an outstanding factor, accounting for no less than 63 per cent of the failures in his analysis. He has also pointed out that cracked and depressed nipples are a formidable obstacle to successful nursing. Although I can quote no figures on these factors, I am convinced that these were the two outstanding difficulties in my own cases. Whose duty is it to care for them? The obstetrician's? The pediatrician's? The trouble seems to lie primarily in the division of responsibility, as suggested above.

The early failure to nurse is largely a psychologic problem, in Pooler's opinion, and it is noteworthy that he finds the idle and uncaring, the stolid and phlegmatic mother less likely to fail in the matter of nursing than the careful and troubled mother, who is often nervous and high-strung. He says, "It is the highest type of womanhood who needs all the help we can give her in the exercise of her maternal duties." This seems to me a very wise statement.

The reports of certain other investigators have been gathered together in Table IV for comparison with our figures.

It is with no pride in our achievements or in these mothers that we record such figures. Yet where does the fault lie? Should we institute a breast feeding campaign, and try again to teach the manual expression of the breasts as advocated by Sedgwick? I confess that the desultory attempts we have made have been attended with very little success. Most mothers refuse to employ the method. Our experience agrees with that of Garland,[4] who states that in Boston and its vicinity he encountered in increasing numbers mothers who were tempermentally incapable of nursing their young for any material length of time, and in still greater numbers those who seemed physically incapable of it even after a conscientious effort had been made.

SUMMARY

1. The superiority of breast feeding is admitted. Disparagement of substitute feeding is a mistake, however.

2. The incidence of breast feeding in 437 mothers is presented. The figures show not only a low percentage of breast feeding during the first month, but a reduction of the percentage in an approximate ratio of one-half, one-third, one-fourth, in succeeding three-month periods.

3. A scoring system for nursing mothers is presented.

4. Age and parity seem to be unimportant factors. Other factors are discussed.

5. Comparison with the findings of others shows no cause for pride in the original series presented.

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636 CHURCH STREET

A CASE OF VON GIERKE'S DISEASE WITH MARKED LIPEMIA

PAUL HOGG, M.D., AND J. BUREN SIDBURY, M.D.
WILMINGTON, N. C.

HEPATONEPHROMEGALIA glycogenica was first described in 1929 by von Gierke. He reported two cases, each of which came to autopsy and revealed a very large liver and enlarged kidneys, without enlargement of the spleen. On microscopic examination he found large quantities of glycogen stored in both the liver and kidneys.

Since von Gierke reported his two patients, about nine cases (to our knowledge) have been discussed in the literature. Practically all of

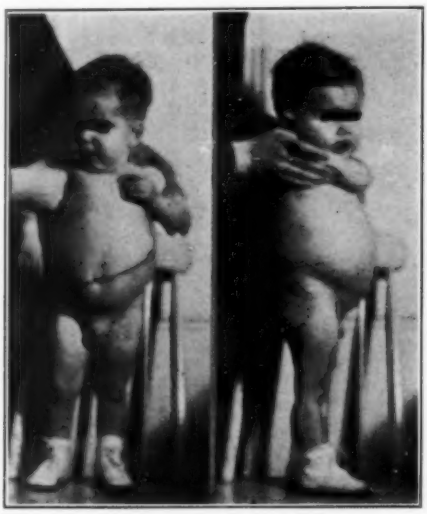

Fig. 1. Fig. 2.

them dealt with faulty glycogen metabolism, but the laboratory findings have varied in most of them. We feel that the physical and laboratory findings of our patient present a rather typical case of this condition.

CASE REPORT

S. N. McK. was first seen at the age of six weeks. At that time he presented a feeding problem, having gained only 6 ounces in six weeks. The family history revealed the father and mother both young and healthy. They gave negative histories for tuberculosis, syphilis, allergy, or any condition that might simulate the patient's trouble. The patient is the only child. He was born at full term; delivery was

From the Babies' Hospital, Wilmington, N. C.

normal; birth weight was 8 pounds and 10 ounces. The baby was breast fed and given complementary feedings of a formula of diluted lactic acid milk. Cod liver oil and orange juice were started when he was one month old, cereals at five months, vegetables at six months, and a three-meals-a-day diet at fourteen months. He had occasional attacks of diarrhea and vomiting which were treated by omitting everything from his diet except skimmed lactic acid milk. At the age of eleven months he had a purulent otitis media that drained for several weeks. About that time he presented an eczema that was caused by the ingestion of egg. When egg was omitted from his diet, the eczema cleared. The baby began to sit alone at the age of nine months and up until the present time he has not walked. The mother first noticed when the baby was two months of age that the abdomen was unusually large. Since then the enlargement has been progressive.

Physical examination at the present time revealed a male child (sixteen months of age) cheerful and apparently in good health, with a markedly enlarged abdomen; height 29½ inches and weight 22 pounds 11 ounces. The head measured 19½ inches in circumference and had a prominent brow similar to that seen in rickets. The

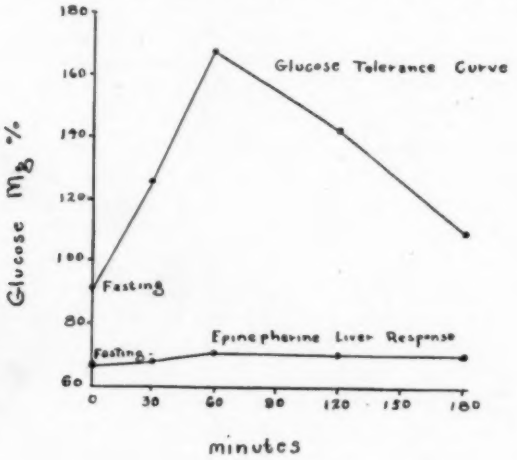

Chart 1.

anterior fontanel was about two fingerbreadths open. The neck was slightly shortened. The eyes, ears, nose, and throat were normal. The eyegrounds were normal. The heart appeared normal to percussion. The heart rhythm was regular and the lungs were normal. The lymph glands were not enlarged. The abdomen was the most outstanding physical finding, being unusually large, measuring 21 inches at the umbilicus. The liver extended nearly to the level of the iliac crest on the right side and to the level of the umbilicus on the left. Dullness was elicited to the fifth intercostal space on the right. The liver was smooth and not tender. The spleen was not felt. There were no signs of ascites or jaundice. The extremities were small and out of proportion to his body. The muscles were flabby.

Laboratory Findings.—The blood study revealed hemoglobin, 85 per cent; red blood cells, 4,000,000; white blood cells, 13,000; differential—85 per cent lymphocytes and 15 per cent polymorphonuclear cells; bleeding time, three minutes; clotting time, three minutes; blood platelets, 400,000. The smear did not show any abnormal cells. The urine had been consistently negative except for a constant acetonuria ranging from one-plus to three-plus. The fasting blood sugar readings

were 93, 71, and 66 mg. per 100 c.c. One and one-half grams of glucose per kilogram of body weight were given by mouth. A normal glucose tolerance curve was obtained (Chart 1). Four-tenths of a 1 to 1,000 solution of epinephrine was given subcutaneously with a very slight rise in the blood sugar. The fasting sugar was 66 mg. per 100 c.c., and during the following three hours the blood sugar did not go over 71 mg. per 100 c.c. (Chart 1). A fasting blood serum was somewhat cloudy but did not suggest the marked lipemia that was revealed by chemical analysis.

	PATIENT	NORMAL
Total lipids	2.770 gm. %	0.570 to 0.720 gm. %
Total cholesterol	0.420 gm. %	0.100 to 0.230 gm. %
Lipoid phosphorus	33.3 mg. %	7.0 to 14.0 mg. %
Neutral fat	1.550 gm. %	0.0 to 0.200 gm. %

(Normal readings from Peters and Van Slyke. Fat determinations by courtesy of Dr. L. E. Holt, Jr., Baltimore.)

Roentgenograms of the skull, long bones, and wrist showed no abnormalities. A flat radiograph of the abdomen showed a large mass that encroached on the thoracic cavity. A barium enema revealed a normal colon. An x-ray picture of the kidneys did not show any noticeable enlargement. A roentgenogram of the heart showed some enlargement, measuring 7.25 cm. in breadth while the transverse diameter of the chest measured 12 cm.

Differential Diagnosis.—In making a diagnosis of von Gierke's disease we considered fatty infiltration of the liver, echinococcus cyst, neuroblastoma of the adrenal gland with metastasis to the liver, congenital cystic liver, and hemangioma of the liver. Kramer's case of fatty infiltration did not have the function of glycogenesis, while our patient can apparently store glycogen in the liver. The complement fixation test for echinococcus cyst was negative. Neuroblastoma of the adrenal gland, cystic liver, and hemangioma of the liver are not accompanied by a hypoglycemia, acetonuria, and lipemia. The large head and large fontanel suggested rickets, but there were no rachitic changes of the bones, especially of the wrist. The negative Wassermann and Mantoux reactions made syphilis and tuberculosis improbable.

DISCUSSION

The most outstanding features of this case are the enlarged liver without an enlarged spleen, the hypoglycemia, aglycogenolysis, acetonuria, retarded physical development, and marked lipemia. This condition apparently deals with a faulty glycogen metabolism. The liver has the power to perform glycogenesis but not glycogenolysis. Schonheimer found the lack of the glycolytic ferment, amylase, in the liver. He believed this was the cause of aglycogenolysis with a resulting increase of glycogen in the liver. Ubshelm found an increase of amylase in the liver and after adding amylase from another liver, glycogenolysis occurred. He thought there was a chemical union between glycogen and protein which inhibited glycogenolysis. Rauh and Zelson found an increase of the diastatic ferment in the urine of their patient, and they believe that there may be an alteration of cell per-

meability. The marked lipemia found in this case is unusual. It is a masked lipemia since the serum on gross inspection did not suggest such high readings. The masked lipemia is suggestive of liver disease. Most likely, the faulty carbohydrate metabolism accounts for the lipemia.

SUMMARY

A case of von Gierke's disease occurring in a sixteen-month-old infant* is presented. Hepatomegaly, hypoglycemia, acetonuria, aglycogenolysis, and marked lipemia are the diagnostic findings.

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*Since preparing this report, the patient has had a generalized convulsion occurring at 7:00 A.M. Physical examination was negative. His temperature was 97° F., rectally. The blood sugar at that time was 56 mg. per 100 c.c.

A "RAPID" MICRO-HINTON AND CAPILLARY HINTON TEST FOR SYPHILIS

With a Discussion of the Detection of Syphilis by Serologic Methods

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ADEQUATE prenatal treatment of syphilitic mothers undoubtedly reduces the incidence of congenital syphilis.[1] Oftentimes, despite inadequate treatment, or even without prenatal treatment, the question arises as to whether the infant has escaped the disease. Manifest stigmas of congenital syphilis may be wanting in the living infant at birth. They may appear after two or three months, but are sometimes postponed until later in childhood or, so far as we know, may never appear. If the serious consequences of congenital syphilis are to be prevented, early detection and adequate treatment are imperative. The fact that an effective specific treatment is available should spur the efforts of physicians to detect these unfortunates in early infancy.

Statistics show that the amount, nature and time of treatment of the mother and the interval between her primary infection and the birth of the child affect the probability of transmission of syphilis. Such figures, however, do not suffice for the individual infant in question. Important as early treatment is, the subjection of all infants to specific therapy simply on the probability of infection is to treat a large number unnecessarily and to place on them the stigma of a disease from which they actually are free. Under no circumstances does the treatment of the newborn infant, or of any child for that matter, seem justifiable until the diagnosis has been established beyond a reasonable doubt. On the other hand, simply to await clinical signs of the disease is to run the risk of irreparable damage to the child.

In the absence of outspoken clinical evidence of syphilis, resort must be made to laboratory aids in diagnosis. Examination of the placenta may give a clue. The demonstration of *T. pallidum* by dark-field examination in scrapings from the umbilical vein[2] or from suspicious lesions sometimes makes an immediate diagnosis of the disease possible, but this method is rather cumbersome. X-ray films of the long bones, when positive, are of great value in establishing the diag-

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The Commonwealth Fund defrayed part of the expense of the study.

nosis. Their usefulness diminishes steadily after the first six or eight months of infancy, and they are often negative in patients who are actually harboring the organisms. Furthermore, instances arise when even the expert eye has difficulty in arriving at an unequivocal decision, and to demand repeated x-rays is to put the individual or clinic to considerable expense.

During the neonatal "period of doubt," including therein the first few months of the lives of infants born of syphilitic mothers, many physicians have come to consider blood tests an uncertain guide. Even with the best methods available at the present time, undoubted cases do arise in the first few weeks of life when false positive reactions are encountered (Case 1). These reactions usually fade out completely within two weeks, although they may last for two or three months.[3, 4] Presumably they are due to the passage of specific reacting substances from the serologically positive mothers through the placentas. A series of titrations of the strength of the false reaction will usually show a progressive decline, but it must be emphasized that the disappearance of this early pseudopositive reaction does not guarantee freedom from syphilitic infection (Case 2).

Another reason sometimes adduced for failure to test the blood of an infant suspected of having syphilis is the observation that occasionally an infected infant fails to develop a true positive reaction, even by one of the more sensitive tests, until several weeks of age (Case 3). This is probably more common in the case of asymptomatic infection, since experience at the Infants' and Children's Hospitals has shown that when clinical signs are present in infancy the Hinton test is rarely negative. It is theoretically possible that some infants with congenital syphilis will never develop either clinical signs or positive blood tests, but such cases are difficult to prove. Indeed, Veeder and Jeans[5] state, "There is no other form of syphilitic infection which gives a higher percentage of positive reactions or in which the reaction is of more value from the standpoint of diagnosis." According to them, over 95 per cent of untreated infants have positive Wassermann reactions and, of those over two months of age, practically 100 per cent are serologically positive. The exact time at which a syphilitic infant will develop a positive reaction depends probably on a number of factors, among them his "serologic immaturity,"[6] the degree and time of his intrauterine infection, and the sensitivity of the test employed.

CASE 1.—White male infant, born at term at the Boston Lying-In Hospital. Birth weight was 7 pounds 10 ounces; length, 20½ inches. The placenta was negative. During the period of observation, the infant developed no signs of congenital syphilis. Although his mother, a primipara, aged twenty-four years, was thought to have congenital syphilis, she was given 15 intravenous injections of neoarsphenamine

during her pregnancy. Two weeks before delivery she developed jaundice and treatment was stopped. Her capillary Hinton test was strongly positive (four-plus). The blood tests on her infant illustrate the disappearance of the pseudopositive reaction.

AGE	CAPILLARY HINTON TEST	HINTON TEST	WASSERMANN TEST
1 day	+ +		Positive
2 days	+ + +		
5 days	+ + +	Negative	Positive
9 days	+		
4 months	-*		
6 months	-*		
13 months	-*		

*Negative reaction.

CASE 2.—A white female infant was born by cesarean section at the Boston Lying-In Hospital, probably a few weeks before term. Birth weight was 4 pounds 8 ounces; length, 19 inches. The placenta was negative, and there were no clinical signs of congenital syphilis in the infant. X-ray films of the long bones were equivocal. The Hinton test for syphilis was negative on the fourth day, and the capillary Hinton tests were as follows:

AGE IN DAYS	RESULTS OF CAPILLARY HINTON TEST
2	+ +
4	+ +
10	+
13	-*

*Negative reaction.

The mother, aged twenty-five years, had had one other pregnancy five years before the birth of this infant, resulting in a miscarriage at three months. Except for two intravenous injections of 0.45 gm. of neoarsphenamine in the last month of this pregnancy, she had had no antisyphilitic treatment. Her capillary Hinton test was strongly positive (four-plus).

The infant gained slowly but steadily, weighing 5 pounds 2½ ounces on discharge from the hospital at two weeks of age. Efforts to secure the return of the infant for periodic examinations were unsuccessful until she was three and one-half months old, when her blood on two occasions one week apart gave positive Hinton and Wassermann reactions at another clinic. Physical examination was negative, but antisyphilitic treatment was begun. Five months later her Hinton test was doubtful and her Wassermann reaction was negative.

CASE 3.—A female, white, full-term infant, born at home, weighing 5 pounds 8 ounces, was admitted to the Infants' Hospital three days later because of hyperpyrexia (105.5° F.) and convulsive twitchings. Her symptoms were thought to be due to dehydration, excessively hot weather and too many blankets. The liver edge was 1.5 cm. below the costal margin, but the spleen was not felt. The cord stump was dry. On discharge, six days later, the capillary Hinton test and x-ray films of the long bones were negative, and she was in good condition.

Her mother, a primipara, aged twenty-two years, with asymptomatic syphilis, had had 0.5 gm. of neoarsphenamine during the last month of pregnancy. Between the ages of seventeen and twenty, a local physician had given her about 24 intravenous injections upon discovering secondary lesions on the face and body. He reported two negative Wassermann reactions at the end of his treatment, but her Hinton test was positive when she applied for admission to the prenatal clinic.

The infant was not seen again by a physician until she was seven weeks old. At that time she returned to the hospital in a poor state of nutrition. She had snuffles and an enlarged spleen. Her Hinton and capillary Hinton tests were strongly positive, but her spinal fluid Wassermann reaction and x-ray films of her long bones were negative. Antisyphilitic treatment was begun at once, with satisfactory clinical response. Roentgenograms of her long bones taken at three months of age showed slight periostitis of the left ulna. At five months of age the capillary Hinton test was still strongly positive.

This uncertainty during the early, neonatal period should not result in complete abandonment of serologic methods of detection. On the contrary, because of the vital importance of early diagnosis and treatment, the situation demands repeated blood and other appropriate tests, in conjunction with careful clinical observation of the infant, until a decision is reached. There appears to be no valid reason for failure on the part of a physician to test the blood of an infant or child on first suspicion of congenital syphilis, at any age.

The time required for venipuncture, the temporary interruption of friendly relations with the patient, objections by parents, and especially the actual difficulties sometimes encountered in withdrawing blood from the vein by needle and syringe all militate against the serologic examination of every infant and child suspected of syphilis. Undoubtedly such patients deserve at least one blood test in addition to indispensable clinical observation. To lower these practical obstacles to routine blood tests, there are at hand a number of serologic methods which require small amounts of serum, such as can be obtained by pricking the finger or toe. Among these are the micromethods of Kahn,[7] Kline,[8] Chediak,[9] and Peterman.[10]

The author has recently developed a micro-Hinton and a capillary Hinton flocculation test,[11] which employ small amounts of serum. The blood is obtained by skin puncture with a No. 11 Bard-Parker blade (V-shaped) and collected either in a long Wright tube or in a capillary tube.* The micro-Hinton test requires 0.2 c.c. of serum and the capillary Hinton test only 0.05 c.c. The regular Hinton antigen or "indicator" is used and the general principles of the procedure are those of the Hinton test,[12] except that after incubation the mixture is centrifuged, so that the positive aggregates are concentrated at the surface of the meniscus. In the case of the capillary Hinton test, readings are made under the low power of a microscope.

Since February, 1935, over five hundred blood specimens, obtained in most instances in a routine way by skin puncture of patients at the Infants' and Children's Hospitals, have been examined by either the micro-Hinton or the capillary Hinton method or both. Recently the capillary Hinton method has been gaining in favor chiefly because it requires a smaller amount of serum (0.05 c.c. or less) and the blood

*11.5 cm. in length by 1.25 to 1.5 mm. in inside diameter. Two shorter tubes may be substituted for convenience in handling.

appears to hemolyze less readily in the capillary tubes. It has been possible to check the accuracy of 464 of these tests against the clinical diagnoses and the results of other tests. Table I summarizes the comparison.

TABLE I

COMPARISON OF RESULTS OF MICRO-HINTON AND CAPILLARY HINTON TESTS WITH THE CLINICAL DIAGNOSES AND RESULTS OF OTHER TESTS

	NEGATIVE	POSITIVE	DOUBTFUL	TOTAL
Micro-Hinton and capillary tests	383	73	8	464
Clinical agreement	382	68	3*	453
Clinical disagreement	1†	5‡	5§	11
Agreement with other tests**	154	68	2	224
Disagreement with other tests**	1‖	4‡	2¶	7

*Cases under treatment for syphilis.

†Patient six days of age. Micro-Hinton positive at seven weeks. Congenital syphilis.

‡Patients with pneumonia, scarlet fever, lung abscess, phlyctenular conjunctivitis, and ratbite fever. The last gave a positive Hinton reaction as well.

§Two patients with deferred diagnoses, three nonsyphilitic patients.

‖Micro-Hinton proved correct.

¶Hinton test negative.

**Wassermann, Kahn, regular Hinton tests, or, in infants under ten months of age, x-ray examination of long bones.

Analysis of the results reveals the fact that recognized cases of false negative reactions were rare and that only five false positive reactions were encountered. The latter were associated with febrile illnesses and four of the five were subsequently retested by the same method and found negative. Of these false positive reactions, only the blood from the case of ratbite fever (a spirochetal disease) gave a strong reaction. This remained so for several weeks.[13]

The accuracy of these tests is further supported by the results[14] of a recent survey of the serodiagnostic tests for syphilis in use in state, municipal and private laboratories, under the auspices of the American Society of Clinical Pathologists and the U. S. Public Health Service. The micro-Hinton and the capillary Hinton tests were found to be 91.9 per cent sensitive and 100 per cent specific. The control Hinton test, performed on the same specimens in another laboratory, was 88.6 per cent sensitive and 100 per cent specific. These results compare favorably with the results obtained by the other participating laboratories and with those of the first evaluation studies under the same auspices one year previously,[15] in which the regular Hinton test was 86.6 per cent, the Kahn test 80.5 per cent, and the Kline test 76.3 per cent sensitive.

With a convenient microtest at hand, blood can be secured with very little ceremony. This appears to be of great practical importance. The ease and economy with which the clinician can obtain blood specimens undoubtedly determines in an appreciable measure the frequency with which these tests are made and, consequently, the

frequency with which cases of syphilitic infection will be detected which otherwise would remain unrecognized. In addition, the micro-Hinton and the capillary Hinton tests share the general advantages of the regular Hinton flocculation test in accuracy, ease of performance,* economy, and stability of the glycerinated antigen or "indicator" employed. Once prepared, this antigen may be kept in the refrigerator ready for immediate use for at least one month, without demonstrable deterioration.

Up to the present, however, the regular Hinton test and the micro-Hinton and the capillary Hinton tests as usually carried out have required an incubation period of sixteen hours. Occasions arise when clinicians desire a serologic report within a much shorter time. This is particularly true when preparations are being made for blood transfusion. Hinton states[16] that his "quick method" gives results only 80 per cent as sensitive as by the regular method. In the original description of the micro-Hinton and the capillary Hinton methods,[11] the author made brief mention of the satisfactory results obtained when the tests were carried out in one hour. Analysis of a longer series is now possible. No apparent loss of sensitivity has followed the shortening of the incubation period to one-half hour from sixteen hours, as will be seen in Table II.

TABLE II

COMPARISON OF THE RESULTS OF THE RAPID MICRO-HINTON AND CAPILLARY HINTON TESTS WITH 238 REGULAR HINTON TESTS

		REGULAR (16-HOUR) HINTON		
		POSITIVE	NEGATIVE	DOUBTFUL
Rapid micro-Hinton	Positive	83		1
(0.2 c.c. serum)	Negative		24	
Rapid capillary Hinton	Positive	61	1	
(0.05 c.c. serum)	Negative		67	1

In other words, 238 "rapid" micro-Hinton and capillary Hinton tests agreed with the regular Hinton tests in all except three instances. One micro-Hinton test was positive when the Hinton test was doubtful; one capillary Hinton test was negative when the Hinton test was doubtful; and one capillary Hinton test was positive when the regular Hinton test was negative.

SUMMARY

The possibilities and the limitations of the serologic detection of congenital syphilis are discussed. The author presents the micro-Hinton and the capillary Hinton flocculation tests, developed especially for pediatric practice. Blood for these tests can readily be obtained by

*Some loss of time in the laboratory results from handling the smaller amounts of serum.

skin puncture and the results appear to be reliable. When the occasion demands, final reading may be made in one hour, without demonstrable loss of sensitivity.

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HEMOPHILIA IN NEGROES

Three Cases and Two Genealogic Charts

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HEMOPHILIA in negroes is exceedingly rare. Bullock and Fildes[1] in 1911 found reports of only three cases in negroes,[2, 3, 4] but did not consider any of them to be authentic. Since then, Crandall[5] has added only one case and believes his patient to be the only instance of true hemophilia in a full-blooded negro. However, he states that the family history in his patient "cannot be interpreted as the genealogy of a hemophiliac."

Fig. 1.—Wound after bleeding had ceased.

Three colored hemophiliacs have been seen at Duke Hospital. Two of them have a definite family history and a genealogy which is fairly typical.[1, 6] In the third case, the diagnosis may be questioned, but the history and x-ray findings are typical of hemophilia.

Case 1.—M. R., No. 77986, a six-year-old male negro, was admitted to Duke Hospital on Dec. 26, 1936, complaining of bleeding from a wound on the head for the preceding seven days (Fig. 1).

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His family history is best illustrated by the accompanying genealogic chart (Fig. 2).

Since the age of one year, the child has been known to bruise very easily. He has had numerous severe nosebleeds, and, when cut, he bleeds profusely and for a long period. Three weeks prior to admission (Dec. 4, 1936) he bruised his forehead. Shortly thereafter a large subcutaneous hematoma appeared. This increased in size

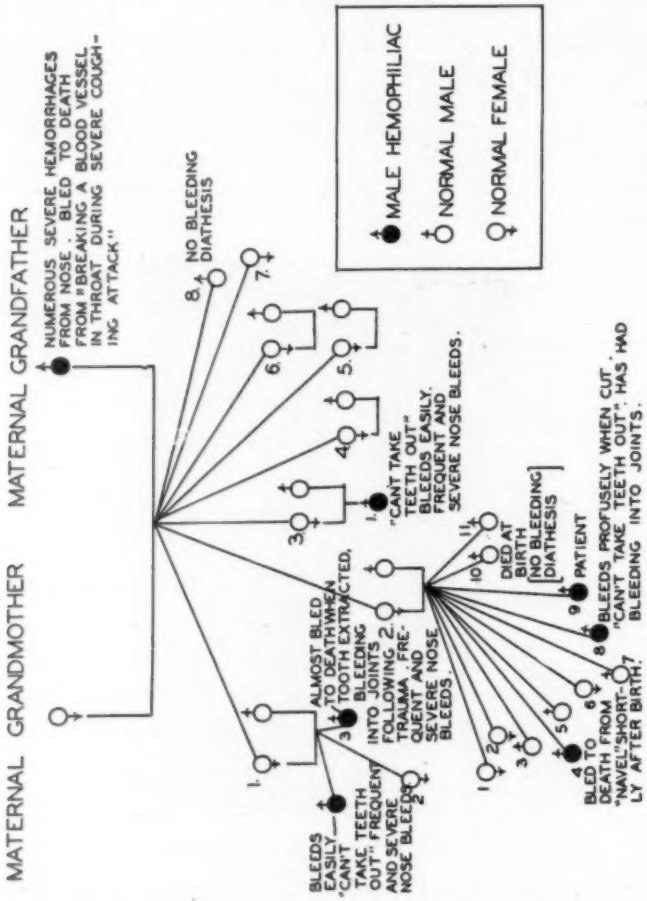
MATERNAL GRANDMOTHER
MATERNAL GRANDFATHER
NUMEROUS SEVERE HEMORRHAGES FROM NOSE, BLED TO DEATH FROM "BREAKING A BLOOD VESSEL IN THROAT DURING SEVERE COUGHING ATTACK"
NO BLEEDING DIATHESIS
MALE HEMOPHILIAC
NORMAL MALE
NORMAL FEMALE
"CAN'T TAKE TEETH OUT" BLEEDS EASILY. FREQUENT AND SEVERE NOSE BLEEDS.
DIED AT BIRTH [NO BLEEDING DIATHESIS]
PATIENT
BLEEDS PROFUSELY WHEN CUT. "CAN'T TAKE TEETH OUT". HAS HAD BLEEDING INTO JOINTS.
BLED TO DEATH FROM "NAVEL STUMP" 7 DAYS AFTER BIRTH.
ALMOST BLED TO DEATH WHEN TOOTH EXTRACTED. BLEEDING INTO JOINTS FOLLOWING TRAUMA. FREQUENT AND SEVERE NOSE BLEEDS.
BLEEDS EASILY—"CAN'T TAKE TEETH OUT". FREQUENT AND SEVERE NOSE BLEEDS.

Fig. 2.—Genealogic chart of first patient (M. R., No. 77986).

for a few days, then became smaller. Six days after the accident (Dec. 10, 1936) fever accompanied by vomiting occurred. He was taken to a physician who believed the fever to be due to the swelling and incised it. The child bled profusely from the wound and continued to bleed after admittance to a local hospital. He was then referred to this hospital for treatment, sixteen days after the incision was made.

On physical examination the patient was a well-developed, well-nourished colored boy who appeared very weak and very fretful. The temperature was 38.8° C. (102.1° F.); pulse, 130; and respirations, 32. There was a large gaping wound

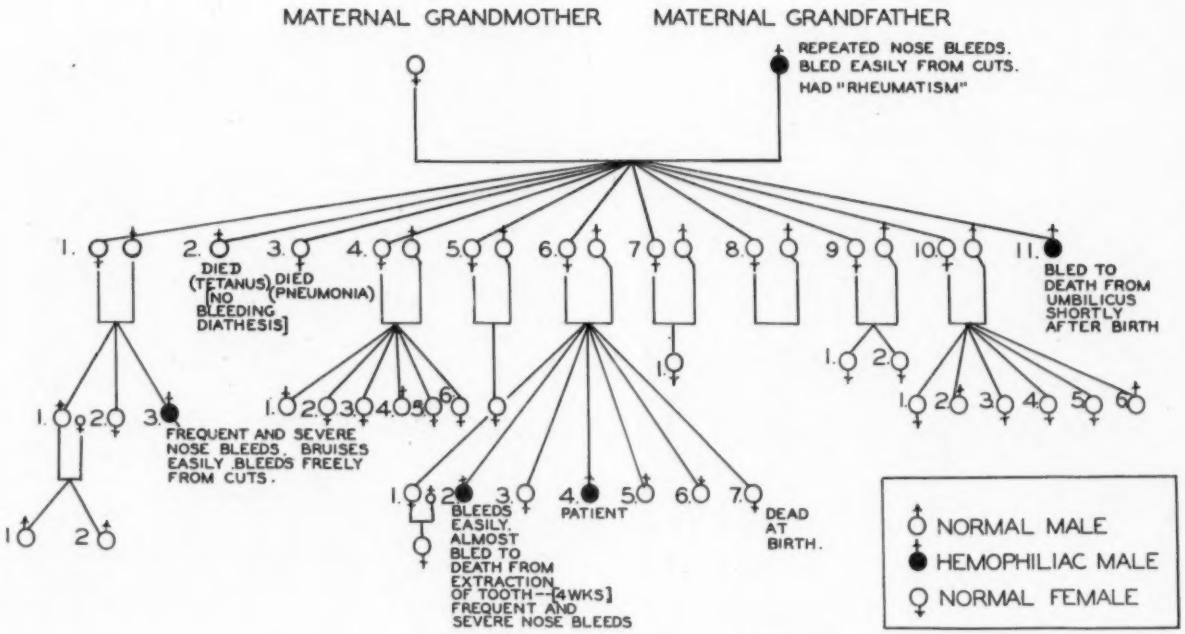
MATERNAL GRANDMOTHER
MATERNAL GRANDFATHER
REPEATED NOSE BLEEDS. BLED EASILY FROM CUTS. HAD "RHEUMATISM"
DIED (TETANUS) [NO BLEEDING DIATHESIS]
DIED (PNEUMONIA)
BLED TO DEATH FROM UMBILICUS SHORTLY AFTER BIRTH
FREQUENT AND SEVERE NOSE BLEEDS. BRUISES EASILY. BLEEDS FREELY FROM CUTS.
BLEEDS EASILY. ALMOST BLED TO DEATH FROM EXTRACTION OF TOOTH—[4 WKS] FREQUENT AND SEVERE NOSE BLEEDS
PATIENT
DEAD AT BIRTH.
NORMAL MALE
HEMOPHILIAC MALE
NORMAL FEMALE

Fig. 3.—Genealogic chart of second patient (G. S., No. 71605).

measuring 5 × 8 cm. (2½ × 3 in.) directly over the left eye, from the base of which blood was oozing in a steady stream. Except for a swollen left eyelid, the results of the physical examination were negative.

Accessory Clinical Findings.—The hemoglobin level was 7.1 gm. (46 per cent); red blood count, 1,600,000; urine, negative. After transfusions calcium was 9.2 mg. per cent; phosphorus, 5.2 mg. per cent; serum proteins, 6.7 gm. per cent; albumin, 3.8 gm. per cent; globulin, 2.9 gm. per cent; albumin-globulin ratio, 1.3. Wassermann and tourniquet tests were negative. There was no sickling of the red blood cells.

Treatment.—The wound was cleaned and some devitalized tissue removed. A pressure bandage was applied but the bleeding continued. He was given a transfusion of 185 c.c. of citrated blood with no immediate effect on the bleeding. On the day after admission (Dec. 27, 1936) the capillary coagulation time was ten minutes, venous coagulation time fifteen minutes, bleeding time three minutes, platelet count 250,000. He received 20 c.c. of human plasma intramuscularly followed by another transfusion of 250 c.c. of citrated blood. Three days after admission (Dec. 29, 1936), following a third transfusion of 125 c.c. of citrated blood, the bleeding ceased. He was subsequently given two more transfusions of 125 c.c. citrated blood each on the fourth and fifth hospital days. The wound began to heal rapidly. Ten days after admission (Jan. 5, 1937), 5 c.c. of blood coagulant extract from the placenta[7] was given orally, and the coagulation time was reduced from fifteen minutes to five minutes within a period of fifteen minutes. The capillary coagulation time remained below seven minutes during the remainder of his hospital stay, and on discharge nineteen days after admission (Jan. 14, 1937), the wound had entirely healed.

CASE 2.—G. S., No. 71605, a thirteen-year-old negro male, was seen in the Nose and Throat Clinic on Aug. 12, 1936, because of frequent attacks of sore throats and of repeated bleeding.

The family history revealed the fact that there are other bleeders in the family (Fig. 3).

The child has been a known bleeder since infancy. Shortly after birth he began to bleed from the umbilicus, and the bleeding was stopped with much difficulty. He has had frequent severe hemorrhages from the nose, bleeds profusely when cut, and has had large subcutaneous swellings when bruised. For the past few years he has had recurrent attacks of sore throat during the winter months. These attacks were usually accompanied by hoarseness and glandular enlargement.

Except for slight deviation of the nasal septum to the right with some injection of the mucous membranes, markedly enlarged and hyperemic tonsils, and hypertrophy of the tissue of the posterior pharyngeal wall, the physical examination was negative.

Because of the family history of hemophilia, the patient was referred to the pediatric clinic. The venous coagulation time was forty minutes plus, and the platelet count and bleeding time were normal. Tonsillectomy and adenoidectomy were thought contraindicated, and x-ray therapy of the posterior pharynx and tonsils was advised.[8] He received six treatments of 160 roentgen units at two-week intervals and the tonsils were reduced in size and the patient stated that he had had no colds or sore throats since x-ray therapy had been started. On Jan. 12, 1937, venous coagulation time was seventeen minutes, bleeding time two and one-half minutes, platelet count normal. The blood calcium was 9.6 mg. per cent; phosphorus, 4.7 mg. per cent; serum protein, 6.68 gm.; albumin, 3.91 gm. per cent; globulin, 2.75 gm. per cent; albumin-globulin ratio 1.4. Ten cubic centimeters of blood coagulant extract were given orally, but this amount apparently had no effect on the coagulation time at the end of thirty minutes. More extensive studies are being done on this patient.

Fig. 4.—Case 3. Hemarthrosis of right knee.

Fig. 5.—Case 3. Anterior posterior views of both knees showing osteoporosis of the left femur and tibia, widening and deepening of the left intercondylar fossa, and arthritic changes.

CASE 3.—F. L., No. 43700, a fifteen-year-old negro male, was admitted to Duke Hospital on Sept. 24, 1934, with the chief complaint of rheumatism.

The family history does not reveal presence of a similar condition in the family. There is no history of hemophilia or other bleeding diathesis in any of the immediate or remote members of the family.

When the patient was about one year of age, it was noticed that he had "rheumatism," characterized by tenderness and great swelling of the knees, elbows, and shoulders. This occurred following trauma; tenderness and painful motion lasted for several weeks. Since that time he has had a tendency to recurrences and has never been entirely free of joint involvement. The present involvement of both knees and the left wrist had been present for three or four months. At the age of three years he fell and cut his eye and lip. He was taken to a physician who had considerable difficulty in stopping the bleeding. On numerous occasions he has had bleeding from the gums. Six years ago, at the age of nine years, a tonsillectomy was performed and was followed by severe hemorrhage, requiring three blood transfusions before bleeding ceased.

Except for a temperature of 39° C. (102.2° F.), enlargement and restriction of motion of the left wrist and the left knee and swelling, tenderness, heat, fluid wave, and restriction of motion of the right knee (Fig. 4), the results of the physical examination were negative.

The hemoglobin on admission was 12 gm. (87 per cent); white blood cells, 7,840; red blood cells, 4,850,000; bleeding time seven minutes and fifteen seconds, coagulation time (capillary method) two minutes and fifteen seconds. Roentgenograms of the right knee showed no bony changes, but there was marked soft tissue swelling and the patellar bursa was distended with fluid, probably blood. The patella was elevated in a manner characteristic of fluid in the joint. In the left knee the x-ray findings were typical of hemophilia with narrowing of the joint spaces due to destruction of the cartilage, erosion of the articular surfaces by old hemorrhage, and deepening of the intercondylar fossa[9, 10] (Fig. 5). Roentgenograms of the wrists and ankles showed only generalized decalcification in the bone. Two days after admission (Sept. 26, 1934), venipuncture in the left antecubital space caused swelling and tenderness of the entire upper arm due to the extravasation of blood. Five days after admission (Sept. 29, 1934), theelin[5] was started in daily doses of 1 c.c. intramuscularly, and continued for five days. Three days after the drug (Oct. 2, 1934) was started, the venous coagulation time was eight minutes (3/8 inch tube); sedimentation rate, normal; the fragility test showed slightly increased resistance of the red cells; platelet count, 380,000; hemoglobin, 9 gm. (58 per cent); bleeding time, twenty-three minutes. The nonprotein nitrogen was 29 mg. per cent, calcium 10.1 mg. per cent; phosphorus, 4.7 mg. per cent; total protein, 7.05 gm. per cent; albumin, 3.31 gm. per cent; globulin, 3.74 gm. per cent; and albumin-globulin ratio, 0.9. The Wassermann reaction was negative.

The patient's condition remained unchanged until the day after theelin therapy was started. The induration in the right knee then seemed to have softened somewhat, and there was less pain on motion. The next day bloody sterile fluid was aspirated from the right knee. Twelve days after admission (Oct. 6, 1934) he was discharged, considerably improved. His subsequent course is unknown.

This patient apparently had hemophilia but Schönlein's purpura cannot be excluded. The history of repeated involvement of the joints, bleeding from the gums, severe hemorrhage following tonsillectomy, profuse bleeding from small wounds is typical of hemophilia. The bleeding time is prolonged in this case, but the coagulation time is also lengthened and the platelet count normal. Aspiration of the enlarged swollen joint revealed the presence of blood and was sterile on culture. Roentgenograms of the involved knee showed changes typical of a hemophilic joint. No information regarding the presence of white blood was obtained from this family.

DISCUSSION

The heredity of hemophilia and its relationship to the sex chromosomes is interesting. Sex is determined by a pair of chromosomes (X) and (Y). The other 23 pairs of chromosomes seem to be alike in both male and female. If two X chromosomes occur, a female results; if X and Y combine, the individual is a male. The hemophilia gene (H) appears only in the (X) chromosome. The accompanying chart illustrates the mode of transmission of the disease (Fig. 6).

X′ denotes a sex chromosome with gene (H). Five possibilities may occur, XY normal male, X′Y male hemophiliac, X′X normal female carrier, XX normal female, and X′X′ hemophilic female. Males resulting

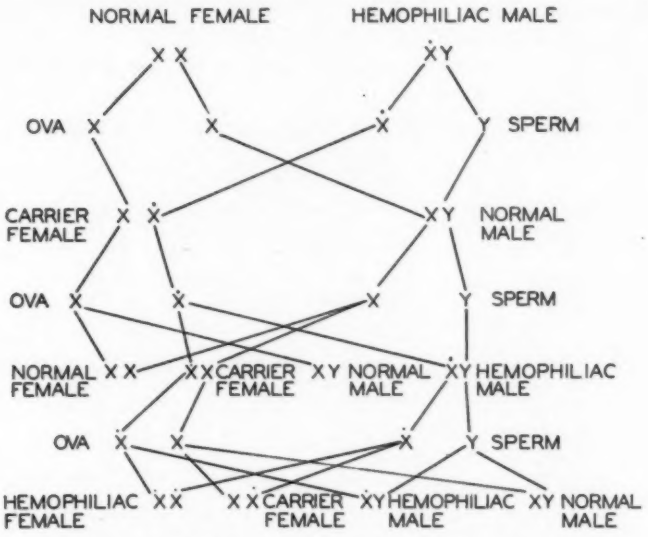

Fig. 6.—Heredity of Hemophilia.
From: Wiener, A. S., Blood Groups and Blood Transfusion, 1935. (Courtesy of Charles C. Thomas, publishers.)

from the union of a hemophilic male (X′Y) with a normal female (XX) will theoretically have normal males, incapable of transmitting the disease. The females, however, will all be potential carriers. Should these females marry normal men, half the sons will be hemophiliacs and half the daughters carriers of the disease.[6] Birch[11] has made the interesting observation in a study of twenty hemophilic families through four to seven generations that the children of persons with hemophilia are more likely to be female than male, whereas the children of these female transmitters are more likely to be males (1.7:1). The heredity in Case 1 closely conforms to the sex-linked theory of inheritance. In the history in Case 2, a male newborn child of the first generation was apparently a

bleeder. The females far outnumbered the males in the first generation in each family, 7:1, 9:2, and the males predominate in the second generation, 8:3, 4:3.

The parents of the patients in Cases 1 and 2 were asked whether white blood was present on either side, but they both denied any interrelationship. The skin and features of Cases 1 and 3 were those of a full-blooded negro, but in Case 2 the color was lighter, indicating that a mixture may have occurred.

The effect of blood coagulant has been tried on two of these cases (Cases 1 and 2) and on other hemophiliacs. This will be reported in another paper.

SUMMARY

1. Three cases of hemophilia in negroes are reported, and in two of them the genealogic charts are fairly typical.

2. Blood coagulant extract from the placenta was tried in two cases, with apparent good results in one case.

3. Theelin therapy was followed by improvement in one case.

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EXTREMITY RESTRAINER SPLINT

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THE LIMB restrainer splint is presented for publication because it has proved to be of practical usefulness in pediatric practice.

The material used is $\frac{3}{16}$ inch duraluminum wire for the small sizes and $\frac{1}{4}$ inch duraluminum wire for the larger sizes. This metal is extremely light in weight and is very rigid. Ordinary aluminum wire lends itself more readily to manipulation, but it does not permit the finished product to be a firm rigid splint. The duraluminum is used extensively in aeroplane construction. The surfaces and edges are read-

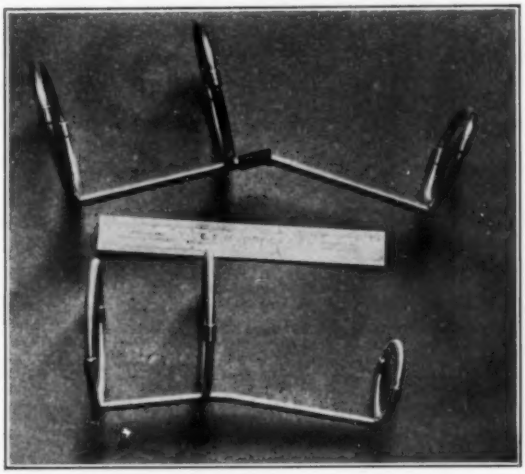

Fig. 1.—Restrainer splints.

ily burnished or polished, leaving a smooth, easily cleaned surface. The splint may be sterilized by boiling. The restrainer consists of three rings attached in parallel to a straight piece. The rings are elliptical in outline and are joined midway by a swivel screw joint. This joint permits the rings to fit the arm closely at three different levels. The rings are closely fitted jointed bracelets. For a baby four months of age, the rings are $1\frac{1}{2}$ by $1\frac{1}{4}$, $2\frac{1}{4}$ by 2, and $2\frac{1}{2}$ by $2\frac{1}{2}$ inches in internal diameters. The proximal ring is almost spherical in outline.

The straight piece is bent slightly or may be hinged to permit 20 degrees of motion. In the small splint, the distances between the rings are 2 inches and $3\frac{1}{4}$ inches. The outside periphery of the ring is

slightly grooved to take the curve of the straight piece, and the attachment is made rigid by use of a staple rivet which passes through the center of the circumference of each ring. In the large splint, the motion in the straight piece is permitted by an asymmetrical slot joint and is covered with a piece of rubber tubing.

The splint prevents, or helps to break, the habit of thumb-sucking in babies and children. Thumb guards do not prevent the child from bringing contamination into the mouth. The more or less constant sucking of the thumb and fingers which have previously been in contact with bacteria-covered surfaces is as important as the expected changes

Fig. 2.—Restrainer splint showing swivel joints.

in the dental arch and tooth direction. Following surgery for plastic repair of extremities, harelip, and cleft palate the restrainer may be of definite help. It is useful to limit motion in eczema, vaccination, and burns.

SUMMARY

1. A restrainer splint is presented because of its practical usefulness.
2. The construction and the material used are described.
3. The uses of the restrainer in some of the more common pediatric diseases are enumerated.

40 Main Street

Critical Reviews

A REVIEW OF AMERICAN RESEARCH ON SEASONAL VARIATION IN STATURE AND BODY WEIGHT

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OVER a century and a half has passed since the first reported observation was published on the relation between the season of the year and growth in stature. Research literature for the interim between 1777 and the present gives evidence of a recurrent interest in the factor of season as related to bodily size and fluctuation in rate of growth. In the United States no less than 23 studies on seasonal variation have appeared to date. It is thus timely that a critical review be made available that will provide a synthetic picture of the current status of American research on this problem.

While the central purpose of this paper is to review American research bearing on the question of whether or not there exists a relation between the season of year and growth in body weight or in total stature, it will be advantageous at various points to articulate American research with the larger body of European research.

The earliest findings on seasonal variation were reported by Buffon[25] in his "Histoire Naturelle" in 1777. This report was based on stature observations taken on a single individual by Montbeillard who measured his oldest son semiannually from the date of his birth in 1759 until he was nearly eighteen years of age. He found no seasonal difference under five years of age. Between the ages of five and ten the stature increments in the summer months were nearly double those in the winter months. Beyond ten years of age the increase continued to be greater in the summer than in the winter but this difference was less marked.

The first reference to seasonal variation in growth published in America was made by a Doctor Nichols in discussing Bowditch's 1872 paper. Nichols stated:

". . . that five-sixths of his own height was due to growth during the summer months, he having been measured every April and September from the age of six months to that of twenty-one years."[6] (P. 435.)

Wretlind[19, 24] who weighed a number of Swedish girls before and after summer vacation, was the first (1878) to find differential weight increments for a group. He attributed the large gain made in the summer months to the effects of the sun and freedom from school but it remained for Malling-Hansen[19, 24] in 1883 to explain the phenomenon as seasonal variation and to substantiate the findings with his own observations on Danish children.

The earliest scientific investigation on seasonal variation in growth in America was reported by Hitchings and Fitz[14] in 1901 and will be

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discussed subsequently in greater detail. In reviewing the American investigations, each one will be placed in one of three groups according to the ages of the subjects studied. The first group deals entirely with newborn infants, the second with infants and preschool children, and the third with children of school age or above.

NEWBORN INFANTS

In 1920 Faber[11] reported the first work on seasonal variation in birth weights in America. His observations extended over a period of thirteen years and included birth weights of 299 boys and 281 girls. These were distributed throughout the calendar year and were obtained at the baby clinic of Stanford University Medical School in San Francisco. Considering each sex separately, the mean birth weight was calculated for each quarter of the year, i.e., January-March, April-June, July-September, and October-December. The results show that there was no seasonal variation in the birth weights of girls but for boys the mean birth weight in the January-March quarter exceeded that in the July-September quarter by 150 grams. The mean birth weight of the six-month period from October to March was 100 grams larger than the mean for the six-month period between April and September.

The next study on birth weights was made by Brenton[7] and appeared in 1922. It was based on more than 2,000 Minnesota birth records taken at the Minnesota General Hospital, the Swedish Hospital, and the University Hospital during the four-year period, 1915-1919. The mean birth weight for each sex was computed for each month of each year and later combined to arrive at an average for each month of the year. Although a slight fluctuation occurred from month to month, no significant or consistent seasonal variation was found for either sex.

Bakwin and Bakwin[1] in 1929 reported the results of a study involving 3,315 birth weights for the years 1926-1927 at Bellevue Hospital, New York. The mean birth weight for each quarter of the year was found but no appreciable difference was noted. In 1934, the same investigators[2] published the results of another study with 1,219 newborn infants as subjects. The mean birth length in addition to mean birth weight was determined for each quarter but no seasonal difference for either measurement was found. The sex of the subjects was not stated in either study.

The birth weights of 4,799 infants from Iowa City, Atlanta, St. Petersburg, Los Angeles, and New Haven were analyzed by Bivings[4] in 1934. Without differentiation as to sex, he found that there was a tendency for birth weights to follow the curve of sunshine—low in fall and winter (mean of 7 pounds 4 ounces) and high in spring and summer (mean of 7 pounds 7 ounces). However, no seasonal variation was noted in the birth weight of the Iowa City infants.

An investigation to determine the influence of seasons on weight loss in infants during the first two weeks of life was made by Bakwin and Bakwin[1] in 1929. The mean weight loss for each month was figured from records of 3,315 infants born at Bellevue Hospital, New York, in 1926-27. It was found that for both sexes the mean weight

loss in the winter months was 8.78 per cent of the birth weight while in the summer months it was 7.5 per cent of the birth weight or 15 per cent less than in the winter months.

Information concerning race and measurement procedure was lacking in all the studies and only in the investigations by Bakwin and Bakwin[1, 2] was socio-economic status of the subjects indicated.

Summary.—The results of the five studies on birth weight and one on total birth length are indicated in Table I.

TABLE I

PERIODS OF MAXIMUM AND MINIMUM BIRTH WEIGHT AS FOUND BY FIVE AMERICAN INVESTIGATORS

INVESTIGATOR		SUBJECT	PERIOD OF MAXIMUM BIRTH WEIGHT	PERIOD OF MINIMUM BIRTH WEIGHT
Faber[11]	1920	299 boys	winter	summer
Faber[11]	1920	281 girls	none	none
Brenton[7]	1922	52 boys	none	none
Brenton[7]	1922	979 girls	none	none
Bakwin and Bakwin[1]	1929	3,315 boys and girls	none	none
Bakwin and Bakwin[2]*	1934	1,219 boys and girls	none	none
Bivings[4]	1934	4,799 infants	spring-summer	fall-winter

*Bakwin also investigated total birth length for this group but found no seasonal variation.

Three of the investigators found no seasonal variation in birth weight for either sex while the fourth investigator found it for boys but not for girls. The study by the fifth investigator showed that birth weights for about 5,000 infants (sex not stated) were larger in spring and summer than those for fall and winter.

In contrast with these results, eleven European studies on approximately 100,000 infants indicate that the period of maximum birth weight occurs in the summer and fall while the minimum occurs in winter and spring. One European investigator found that infants born in the summer were lighter than infants born in the winter while a second investigator found no seasonal variation. The study on seasonal variation in weight loss following birth reveals that in both sexes the loss in the winter months was 15 per cent greater than in the summer months.

INFANCY AND PRESCHOOL YEARS

Seasonal variation in weight gains for the first two years of life was reported by Bleyer[5] in 1917. The data were accumulated over a five-year period prior to 1917 from 3,800 weight records of 1,000 infants at the Well Baby Clinic of Washington University, St. Louis. An average of three weighings at monthly intervals were taken on each subject and none were included who showed signs of illness on the weighing day.

The first-year infants showed the largest gain in weight in the summer months but the second-year infants apparently had two maxima, one in February and March and another in October with a

minimum in June. In considering the results, the fact that the data are discontinuous (subjects not measured for 12 consecutive months) should be recognized. The sex of the infants was not stated.

Faber[11] kept records on 200 breast- and bottle-fed infants and reported that maximum growth of the former group occurred in March and November and for the latter in February and August. Careful inspection of his graphs does not reveal a marked seasonal growth trend since there are numerous fluctuations but it seemed to be slightly the best for gains in both groups in the autumn months.

Berkson[3] in 1930 presented statistical evidence of a seasonal cycle in growth by analyzing the data in Woodbury's report, "Stature and Weight of Children Under Six Years of Age." Means, each based on approximately 15,000 cases, were available for stature and weight for each sex for each month of age below seventy-two. By comparing the theoretical value of the mean for any given month of age as read from a parabolic curve fitted to the means by least squares with the obtained mean for that age, Berkson obtained a statistically significant difference at many ages. In consideration of these results, he concluded that there is a seasonal variation in growth because most of the obtained means above the theoretical means came in one season of the year and those below came in another season. He did not state the season in which the rate of growth appeared to be most rapid.

Wallis[28] computed the mean gain in stature and weight for two periods, November to May and May to November for 120 New York private school children between the ages of two and eight years. Stature was measured without shoes and weight was taken in the nude. The measurements which cover a five-year period, 1926-1930, were taken by the person making the investigation. Each sex was treated separately and the following conclusions were made:

> That time of year does not affect increments of stature and therefore a large proportion of spring or winter measurements in any age group would not emphasize or weaken the expected gain. . . . This is in accord with recent belief that under good environmental conditions there is no real seasonal variation in the amount of a child's growth.[28] (p. 31.)

> No seasonal differences in amount of weight gained were found among the New York children. The increments of weight conform closely to those of stature in illustrating the absence of seasonal influence on the increase of bulk.[28] (p. 37.)

In 1931, the same year in which Wallis' publication appeared, McKay and Brown[18] reported the results of a three-year investigation covering the same age range. Consecutive monthly measurements of height and weight for a period of one year on 114 Ohio preschool children, found by a pediatrician to be in good physical condition, constituted the data for the study. Indoor clothing minus shoes and sweaters was worn when weight was taken and stature was secured with the subject in stocking feet. The measurements were made about the same time of day each month by one investigator and were checked by another.

The mean monthly increments were determined but for purposes of this study the year was divided into two periods of six months each—

May to October and November to April. The same trends were found for each sex. Conclusions from the study were:

> There is a decided tendency toward a seasonal variation in the rate of growth in weight of preschool children, the period of most rapid growth being in the late summer and early fall, with the period of minimum gain in winter and spring.
>
> Seasonal variations in increase in height were so small as to be insignificant. This may indicate that as far as increases in height are concerned, environment is less a factor than is the case with increase in weight.[18] (p. 32.)

Summary.—The two investigations on stature (McKay and Brown, and Wallis) reveal no seasonal variation in growth rate for either sex. The four studies dealing with seasonal variation in weight gains in infants and preschool children are summarized in Table II. It is noted that (1) three investigators found the summer and autumn months most conducive to weight gains, and (2) no seasonal variation in weight increments was found by the fourth investigator. The results of European investigations on this age range are so inconclusive that a comparison would not be profitable.

TABLE II

PERIOD OF MAXIMUM AND MINIMUM WEIGHT INCREMENTS FOR INFANTS AND PRESCHOOL CHILDREN FOUND BY FOUR INVESTIGATORS

INVESTIGATOR		SUBJECT	PERIOD OF MAXIMUM CHANGE	PERIOD OF MINIMUM CHANGE
Faber[11]	1920	200 infants	fall	spring
Bleyer[5]	1917	1,000 infants	summer	late spring
Wallis[28]	1931	about 60 boys	none	none
Wallis[28]	1931	about 60 girls	none	none
McKay and Brown[18]	1931	about 60 boys	summer-fall	winter-spring
McKay and Brown[18]	1931	about 60 girls	summer-fall	winter-spring

SCHOOL AGE OR ABOVE

As previously stated, Hitchings and Fitz[14] published the first quantitative American study on seasonal variation in 1901. The subjects were 20 boys between seven and fourteen years of age at the Dedlam Boys' Home, a branch of the Boston Children's Friend Society. Weight was taken weekly in the nude and height was measured quarterly for an unknown period of years. Findings from the investigation are:

> The variations in weight amounting to as much as five pounds were observed in most of the boys from week to week. The variations were so numerous and so marked as to suggest that any single test of an individual is liable to a plus or minus error of several pounds and that successive weighings are necessary for accuracy.
>
> Over 90 per cent of the total increase in weight occurred during the period from June to December.
>
> During the period from January to June the weight fluctuated without definite gain or loss, constituting a period of minimum growth.
>
> The general form of the curve of growth in weight was found common to all the individuals studied (20), covering the ages seven to fourteen. The larger

> minor fluctuations were also found to be common to the majority of the boys, suggesting that the causes were general rather than individual, and that growth throughout this age period has the same general seasonal variation.
>
> Growth in height in the majority of cases showed either a continuous increase at the same rate throughout the year or more rapid growth during the period of most rapid growth in weight. Only three boys showed correspondence with Malling-Hansen's conclusions, that is, more rapid growth in height during the resting period of growth in weight.[14] (pp. 511, 512.)

The preceding statements, although based on few cases, merit consideration in the planning of any study on seasonal variation in growth.

In 1918, Holt[15] reported the results of a study on seasonal variation in weight and stature. The mean weight increment for two periods, November to May and May to November, was secured for 703 boys ranging in ages from five to sixteen in the Browning School, New York. The average gain was one and one-fourth pounds greater in the May-November period than in the November-May period.

Mean monthly height increments on 544 boys were found to be 0.36 inch larger in the summer months than in the winter months. Holt believed this due to a more active life and lack of colds and diseases in the summer. A description of the measurement conditions was lacking.

A major investigation on seasonal variation in weight was conducted by Porter[22] and extended over an eight-year period. Starting in 1911, Porter secured weight and height on several thousand of the youngest school children in Boston. With numerous exceptions, he was able to observe these two measurements monthly on the same children until 1919. The clothing* worn by the subjects at the time of weighing was shown by Porter to have no effect on the results. For all boys born in 1905, the mean monthly gain in weight from January through May was 0.21 pound and from June through December was 0.77 pound. The same trend was noted in each of the eight years of the investigation. No seasonal variation in stature gain had been found by Porter at the time of publication of the results on weight gain.

A study of seasonal variation in weight and height was made by Hunt, Johnson, and Lincoln[17] on two groups of Russian and Austrian boys in New York City in 1919-20. A nutrition class of 44 boys between the ages of five and seven who averaged 11.6 per cent underweight and an equal number of control boys who averaged 0.83 per cent underweight in September, 1919, were weighed and measured in October, February, June, and October. Both measurements were taken without shoes or coats but no account of the technique employed was given. The results show that the October-February period was most conducive to weight gains for both groups although more marked for the nutrition class. Height increments were greater for both groups in the February-June and June-October periods.

*Turner[25] has since added to the available information concerning the effect of clothing on studies of seasonal variation by making a detailed study on the weight of many garments worn by children while being weighed. This study is helpful where weight is taken with the subject clothed but a more desirable procedure is to secure weight in the nude or with the subject wearing only a robe, the weight of which is known.

Holt and Fales[16] in 1920-22 investigated periodic growth of children in a Catholic institution for the poor. They secured 2,995 monthly weights on 151 boys and 195 girls of French, Italian, Spanish, and Irish parentage. Each subject was weighed from one to twelve times, the weight of the clothes being subtracted from the observed weight in each instance. According to the authors, the general physical condition was above that of children in private homes because they followed a routine schedule of living habits. The results indicate that while wide fluctuations occurred from month to month, little if any seasonal variation in weight occurred for these institutional children. The mean June-to-December gain for 187 girls was 3.26 pounds and the mean December-to-June gain for 144 girls was 3.34 pounds. For 113 boys in the summer months the mean gain was 3.50 pounds while in the winter months for 85 boys it was 2.44 pounds.

Another study on New York City children was carried out by Gebhart.[12] He took consecutive monthly weights on 363 Italian boys and 614 Italian girls between two and twelve years of age for periods of three to five months each. The average period of observation was 3.8 months for boys and 4.5 months for girls. The average monthly gain for each age and sex was computed but because there were so few in each age group the average monthly gains were converted by proper weightings into gains for the nine-year-old group since the greatest number was in this group. The conversion was accomplished by multiplying the monthly gain for each age by the percentage ratio between the average expected annual gain for that age and the average expected annual gain for the nine-year-olds. The expected annual gains below six years of age were taken from Woodbury's figures and those above six years were from Burk-Boas. Weight was taken in indoor clothing but no other account of technique was given. The validity of his procedure of combining short periods of observations to determine the amount of seasonal variation in weight may be questioned. The results show that the greatest gain in weight for both sexes occurred in September, October, and November while the least gain occurred in May and June.

Dividing the year into three equal parts, Veeder and Rohlfing[27] made weight observations in October, January, and May on 534 selected boys between the ages of ten and eighteen at the St. Louis Country Day School. The time of day or order of measurement was not given but weight was taken in the nude and all records of sick, injured, underweight, and overweight boys were excluded. There were between 50 and 97 subjects in each age group except the eighteen-year-old one which had 24. The boys between the ages of ten and fourteen gained weight fairly uniformly throughout the year but from age fifteen to eighteen most of the gain occurred in the October-to-January period of the year. The authors pointed out that the average gain cannot be applied to the individual rigidly since there is a wide range used in obtaining the average.

In 1926, Porter and Baird[23] reported that at certain equal ages in months, two groups of boys, one born in February and the other in August, differed markedly in median weight. The February group was heavier at some ages (107 and 109 months), the August group at other ages (109 and 121 months), while the two were approximately equal at any age in months which was divisible by 12 (72, 84, or 96).

The authors explained this phenomenon on the basis of Porter's[22] findings on seasonal variation in 1920. It was noted that the advantage held by each group appeared at regular intervals depending on the number of good growing months which each had had. The same trends occurred for both sexes for those individuals born between January 1 and June 30 and those born between July 1 and December 31. The number of cases was not given but the results seem to substantiate Porter's previous findings.

Monthly measurements of stature and weight on 833 children for a period of one year were obtained by Emerson.[10] Eighty per cent of these children who represented three geographic localities, New York, Toronto, and Honolulu, made irregular gains in weight. Although no seasonal trend for weight was found by Emerson, he was of the opinion that the regular monthly increment of the remaining 20 per cent was contrary to expectations. Stature gains appeared not to be influenced by seasons. Other than the fact that "measurements were free from any gross error of clothing and posture," nothing is known concerning the procedure or sex.

From 1910 to 1928, Gould[13] secured stature and weight measurements in the fall and spring on 875 college girls for a period of four years each. The mean increase in stature in four years was only 0.33 inch but 0.27 inch of it occurred in the winter months. There was a mean weight increment of less than one pound for the same period but during the winter months a mean decrement of about one pound was noted while a slightly larger increment occurred in the summer months.

Between 1925 and 1929, an unpublished study of seasonal variation in weight of children attending the Kansas State School for the Deaf was made by Chaney and Justin.[8] Three groups of each sex between the ages of six and ten furnished the data for the study. One group received two medium-sized oranges daily in addition to their regular fare, another group received daily treatment from an Alpine Sun Lamp while the third was a control group. With a few exceptions, monthly weights were secured on about 40 boys and 40 girls for the three school years 1926-1929 but for the purpose of this study the school year was broken into two parts—September through December and January through April. Weight was taken at the same hour each observation period with the subject in indoor clothing but without shoes, sweaters, or coats. Each year was considered separately and therefore results were computed on 187 "case years."

The investigators found the gain in weight to be greatest in all groups in the fall months for children between six and nine years of age but no significant difference was noted for ten-year-olds.

An important investigation on weight increments was reported in 1933 by Palmer[20] as part of a health demonstration project at Hagerstown, Maryland. The subjects for this study were 2,500 native-born white children between the ages of six and sixteen who were in attendance at the eight elementary schools of Hagerstown. Weights on the children were secured each school month from September, 1923, to May, 1928. Grades 1, 2, 3, and 4 were used the first year; 2, 3, 4, and 5 the second year, etc. Children with less than 80 per cent of a complete record for four of the five years were not used in the analysis.

One person took all the weights with the subjects wearing only regular indoor clothing without shoes. Palmer pointed out the validity of this procedure since the greatest gains in weight did not occur when heavier garments were worn by the children using them nor did the smallest gain occur when the heavier garments were discarded. The day of the month varied as did the time of day. Palmer felt that the factor of the time of day was negligible and he made corrections for the difference in number of days between weighing periods. The children were placed in one-year age groups using the age of the birthday nearest January first and the exact mean age of each group since this was considered more accurate.

A table was compiled which shows the number of days for each age and sex, the number of cases, and mean gain for each period indicated. There is also a graph which shows the fluctuation in the gain in weight by month for each of the ten age groups used. The procedure employed in making the statistical analysis was carefully described.

Palmer summarized his findings in relation to seasonal variation in weight as follows:

> Maximum rates of average growth in weight are observed during the fall months, intermediate rates during the winter, and minimum rates during the spring. The average rate of growth during the summer period is approximately equal to the rates observed during February and March. The same cyclic changes are observed in both sexes and for each yearly age group from the sixth through the fourteenth year. These findings are in agreement, with few exceptions, with the previous work on the subject.[20] (p. 231.)

Davenport, Steggerda, and Drager[9] in a study of Letchworth Village boys noted that:

> . . . Growth in stature in our series is most rapid in the spring; while growth in weight is most rapid at the same time and also hardly less so in the autumn. There is a physiologic need for autumnal acceleration in weight that hardly exists for stature.[9] (p. 283.)

The last statement of the preceding quotation is taken to mean that the extra weight acts as an added protection against winter cold. The number of subjects and conditions surrounding the measurements were not given for this study.

One of the most recent studies on seasonal variation and also one of the most thoroughly executed was published in 1935 by Whitacre.[29] Observations were made on 2,542 white, Mexican, and negro boys and girls between the ages of five and nineteen in attendance in three San Antonio public schools with division as to race and sex as follows:

RACE	BOYS	GIRLS	TOTAL
White	519	462	981
Negro	362	369	731
Mexican	421	409	830
Total	1,302	1,240	2,542

Weight was taken monthly from January 1929 to April 1931 except in summer vacation. A periodically checked scales was used and each child removed his clothes and wore a robe, the weight of which was

subtracted from the observed weight to give the child's actual weight. Height was taken once a semester or five times in all by means of a Baldwin measuring tape fastened securely to a specially constructed vertical board. All measurements were taken two or more times to insure accuracy.

Individuals were weighed at the same time of day as far as possible and each child went to the toilet to empty the bladder before weighings. The longest interval between weighings during the school year was forty-two days in October 1929 and the shortest was twenty days in November 1929. The number of weights secured each month varied because of changes, absentees, and because a record of loss in weight over the weight at the preceding weighing period due to illness was not used. Weights as great as, or larger than, preceding weights were considered valid and were used even though the child had been ill. Age was figured from January first and was used throughout the calendar year.

The following excerpts from Whitacre's study explain some of the procedure and give the results of the investigation.

> Evidence of seasonal variation of growth in weight was sought on the bases of (1) average net gains per child derived from group weights, (2) comparison of the contribution made by gainers and losers to the net monthly gain, (3) examinations of the individual records of 98 pupils (42 white, 28 each of Mexican and negro) who showed no loss between any two consecutive weighings. The spring season included February, March, and April; the summer period extended from May through September; and fall included October, November, December, and January.[29] (p. 69.)

The effect of seasons on weigh gains for each of the three races is as follows:

> The results in all analyses show that race likenesses are much more pronounced and consistent than race differences. For all three races, fall was slightly the best season for gaining. . . . Among the three races, October was conspicuous for consistently excellent gains (from 11.3 per cent to 16.1 per cent of the yearly gain), and April, for uniformly poor gains (from 1.4 per cent to 6.4 per cent of the yearly gain).
>
> The records of individual monthly weight changes show that all three races have higher ratios of gainers to losers in fall months than in those of spring. . . .
>
> Approximately two-thirds of the individual records of the constant gainers agreed with the trends shown by the monthly weight changes and by the average net gains derived from group weights. . . . Each child should be regarded individually in his course of growth. A child's failure to gain should not be attributed complacently to the season of the year. Too much departure from regularity of gain calls for special attention, although the degree of uniformity to be expected in normal growth must be determined by further study.
>
> Seasonal variation of growth in weight appeared not to be influenced by sex, age, birth-month, type of body build, or living conditions as indicated by the average number of rooms per person in the house and the presence or absence of a bathroom.

No explanation of the excellent gains made in October or the poor gains in April was afforded by examination of the records of weight changes in relation to the records (1) of food eaten for from two to four days within the weighing period, (2) of the minor illnesses, especially colds, or (3) of climatic conditions as indicated by U. S. Weather Bureau records.[29] (pp. 69-71.)

Findings on height increments are:

Average monthly gains in height, both standing and sitting, for both the entire group of each race and age groups of white and negro children show exceedingly small and negligible differences in spring-to-fall and fall-to-spring periods. . . . It appears from the data of this study that growth in height was relatively uniform through the year, or perhaps marked difference in rate was confined to shorter time intervals than those used in this study.[29] (p. 71.)

Summary.—The results of the ten American studies on seasonal variation in weight gains of school children of one or both sexes are shown in Table III. No seasonal variation was reported in two, one

TABLE III

PERIODS OF MAXIMUM AND MINIMUM GAIN IN WEIGHT OF CHILDREN OF SCHOOL AGE OR ABOVE AS FOUND BY ELEVEN INVESTIGATORS

INVESTIGATOR		SUBJECT	PERIOD OF MAXIMUM GAIN	PERIOD OF MINIMUM GAIN
Hitchings and Fitz[14]	1901	20 boys	June-December	January-May
Holt[15]	1918	703 boys	May-November	November-May
Porter[22]	1920	several thousand boys and girls	June-December	January-March
Hunt, Johnson, and Lincoln[17]	1921	90 boys	October-February	June-September
Holt and Fales[16]	1923	113 boys	June-December	December-June
Holt and Fales[16]	1923	187 girls	none	none
Gebhart[12]	1924	363 boys	September-November	May-June
Gebhart[12]	1924	614 girls	September-November	May-June
Veeder and Rohlfing[27]	1927	534 boys	October-January	May-September
Emerson[10]	1927	833 children	none	none
Chaney and Justin[8]	1930	187 boys and girls	September-December	January-April
Palmer[20]	1933	about 1,250 boys	September-December	March-June
Palmer[20]	1933	about 1,250 girls	September-December	March-June
Whitacre[29]	1935	1,302 boys	October-December	April
Whitacre[29]	1935	1,240 girls	October-December	April

on a group of which the sex is not known and the other on a group of girls in an institution. The latter study marks the only sex difference reported—the boys who were observed by the same investigator showed a seasonal variation in weight gains which agrees with the results of the other eight investigations. Fall was found to be the optimum time of year for weight increase with summer second best while winter and spring were least conducive to growth in weight. The three major investigations[20, 22, 29] each based on consecutive measurements of 2,500 children agree on these results as do the majority of European studies.

Four of the six investigations on seasonal variation in stature yielded results indicating an absence of seasonal growth. The other two in-

vestigators reported that the spring and summer months were the most favorable to increase in stature with winter the least favorable. Table IV shows the results.

TABLE IV

PERIODS OF MAXIMUM AND MINIMUM GAIN IN STATURE OF CHILDREN OF SCHOOL AGE OR ABOVE AS FOUND BY SIX INVESTIGATORS

INVESTIGATOR		SUBJECT	PERIOD OF MAXIMUM GAIN	PERIOD OF MINIMUM GAIN
Hitchings and Fitz[14]	1901	20 boys	none	none
Holt[15]	1918	544 boys	summer	winter
Porter[22]	1920	several thousand boys and girls	none	none
Hunt, Johnson, and Lincoln[17]	1920	90 boys	February-October	October-February
Emerson[10]	1927	833 children	none	none
Whitacre[29]	1935	1,302 boys	none	none
Whitacre[29]	1935	1,240 girls	none	none

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PROGRESS OF PUBLIC HEALTH AS IT RELATES TO THE CHILD

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MORBIDITY AND MORTALITY

THE year 1936 produced no nation-wide epidemics of notable proportions and there was on the whole a moderate to low incidence of the acute communicable diseases of childhood.[1] Meningococcus meningitis, which had been on an upward swing for the preceding two years, reached a new height of incidence for this cycle during 1936; the highest rate since 1929. Smallpox continued to show a high incidence in certain sections, particularly in the north central, mountain, and Pacific regions, and this was reflected in a slightly elevated national rate.

Influenza assumed minor epidemic proportions during the early months of 1936, and again became very prevalent during the same months of 1937 in certain sections of the United States. It did not, however, attain the prevalence in this country which was reported in Central and Northwest Europe. Scarlet fever had been on the increase in most sections of the United States during 1935 and this continued well into 1936, but has since declined and remained at a relatively low level. During the fall of 1936 infantile paralysis occurred with a moderately high incidence in the north central and certain other sections, but along the Atlantic coast, where it had been prevalent during the season of 1935, and in the far West the incidence was relatively low. Diphtheria registered an all-time low level during 1936, and measles and typhoid fever have continued at exceptionally low levels of incidence.

The restricted scope of morbidity statistics and their lack of reliability were commented upon in our 1936 review,[2] and the technique of periodic field surveys in sample communities was described as a means of obtaining more information regarding morbidity. The periodic canvasses now being conducted by the United States Public Health Service are designed to gradually build up a body of epidemiologic knowledge not readily obtained in other ways, and especially to provide means of determining the trends in the natural history of diseases and the effectiveness of preventive procedures. Interesting reports based upon these surveys which appeared during 1936 have dealt with the incidence of whooping cough,[3] with the frequency of diphtheria immunizations and cases,[4] and with smallpox vaccinations and cases[5] among the sample populations. Collins, in a study of diphtheria covering the years 1928 to 1931 inclusive, found that the peak of case incidence occurs at about four years of age in the South but not until about six years of age in the North. The peak of mortality occurred at two years of age with the

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maximum case fatality at about one year. The histories showed that attacks were more common among males than females and were most frequent in large cities, and least frequent in rural areas. Seven per cent of individuals twenty to twenty-four years of age gave a history of having had an attack. Forty-three per cent of nine-year-old children gave a history of an artificial diphtheria immunization.

In a study of smallpox during the same period Collins found that the case incidence was highest in the twenty- to twenty-four-year-old group but next highest under one year. Two and seven-tenths per cent of twelve-year-old children gave a history of having had the disease. The case fatality varies greatly from year to year but is again relatively high among infants and young children. The peak of mortality was highest in the age group over sixty-five, but the next highest rate was under five years; a rate between four and five times that among ten- to fourteen-year-old children. Only 23.6 per cent of five-year-old children and 65.9 per cent of twelve-year-old children had ever been vaccinated. Large cities showed an earlier and higher percentage of vaccinations and fewer histories of the disease than small communities and especially than rural areas. Wide variation was found in the percentage of those vaccinated in different sections and communities. In another study dealing with college students[6] Bull found that 33 out of each 1,000 students had never been vaccinated and that 75 of each 1,000 did not have an adequate protective immunity against smallpox. Hedrich[7] has reviewed the changes in recent years in the incidence and fatality of smallpox and considers their relation to vaccination and quarantine.

At the time of submitting this review, the tentative report of mortality in certain states during the year 1936, quoted in previous reviews for the year in question, is not available. Table I, however, presents similar figures covering the first nine months of the year 1936 and the same period in each of the three preceding years. This table has been prepared from figures published by the United States Public Health Service.[8] The figures presented deal with conditions of particular importance to children, although only certain of the figures are limited to an age period. It can be readily ascertained from these tentative figures that in spite of a rise in the total death rate (due apparently primarily to a sharp increase in deaths from all forms of pneumonia and a high influenza rate) both maternal and infant mortality have reached new low levels. Most of the specific rates are also very favorable.

In connection with studies of mortality among children, fatal accidents have been coming more and more into prominence, and several interesting studies of these have been reported during the year by the Public Health Service.[9]

MATERNAL HEALTH

The problem of maternal mortality has been extensively discussed during the past year, and further evidence has been presented indicating that death resulting from pregnancy could be markedly reduced by more wide adoption of accepted obstetrical practices. It is not difficult to account for the increasing interest in this subject since puerperal conditions collectively rank second to tuberculosis as a cause of death among women between the ages of fifteen and forty-four in the United States. They account for approximately one-sixth of all deaths occurring among women in the third decade of life. It is difficult to compare rates for

these causes in different places and in different periods because of the wide divergence of procedure in classification. But the evidence suggests that after a long period of stationary, if not actually rising, rates there has developed during the past five years a definite trend toward gradual reduction.

TABLE I

SUMMARY OF MORTALITY FROM CERTAIN CAUSES IN A GROUP OF STATES FIRST NINE MONTHS OF EACH YEAR 1933-1936

FIGURES OBTAINED FROM THE REPORTS OF THE OFFICE OF STATISTICAL INVESTIGATIONS, UNITED STATES PUBLIC HEALTH SERVICE				
DISEASES (NUMBERS IN PARENTHESES ARE FROM THE INTERNATIONAL LIST OF CAUSES OF DEATH, FOURTH REVISION, 1929)	JAN.-SEPT. 1936	JAN.-SEPT. 1935	JAN.-SEPT. 1934	JAN.-SEPT. 1933
21 states (est. population 71,740,000)*	*Death Rate Per 1,000 Population (Annual Basis)*			
	11.4	10.8	11.0	10.6
	Deaths Under One Year Per 1,000 Live Births			
Total infant mortality	52	53	58	55
All infant mortality except malformation and early infancy	22	23	25	24
Maternal mortality	5.2	5.4	5.7	5.8
	Death Rate Per 100,000 Population (Annual Basis)			
Diarrhea and enteritis under 2 yr. (119)	8.2	7.6	10.5	9.8
Typhoid fever (1, 2)	1.5	1.8	2.3	2.3
Measles (7)	0.7	3.9	4.9	1.9
Whooping cough (9)	1.9	3.8	4.8	2.9
Scarlet fever (8)	2.3	2.4	2.2	2.1
Diphtheria (10)	1.5	1.8	1.8	1.8
Acute anterior poliomyelitis (16)	0.4	0.7	0.6	0.7
Meningococcus meningitis (18)	2.5	2.2	0.9	1.2
Influenza (11)	21.3	20.9	14.8	27.0
Pneumonia, all forms (107-109)	88.9	82.2	80.0	69.3
Tuberculosis, all forms (23-32)	52.4	52.6	53.7	56.9

*States included: Connecticut, District of Columbia, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Louisiana, Maryland, Michigan, Minnesota, Montana, New Jersey, New York, Pennsylvania, South Dakota, Tennessee, Virginia, West Virginia and Wisconsin.

During the year, the Obstetrical Society of Boston and the Boston Department of Health completed a detailed analysis of the causes of maternal deaths occurring in the city during the years 1933, 1934, and 1935.[10] This study followed very much along the lines of studies previously conducted in other cities and with the same general results, chiefly that a number of the deaths were directly traceable to bad obstetrics, while a number of others were traced to negligence on the part of the pregnant woman. Tucker and Benaron[11] have reported on four years' experience at the Chicago Maternity Center, a large out-patient obstetrical service which operates in a poverty-stricken environment. They attribute their surprisingly low maternal mortality to rigid adherence to the following principles:

A trained obstetrician in complete charge.

Adequate prenatal care available, reinforced by field nursing follow-up.

Constant attendance during labor (doctor, medical student, or nurse).

Adequate equipment and personnel for both normal and operative deliveries.

A simple, adequate, and consistent technique followed.

Isolation in the home.

A minimum of operative interference.

Good hospitals available.

Prompt attention to the control of hemorrhage and the replacement of lost blood.

Taboo of pituitrin until after baby is delivered.

Sane use of obstetrical and surgical anesthesia.

The programs which have been instituted in the various states as a result of the Social Security Act are quite generally featuring the development of maternal health services. Institutes affording intensive postgraduate instruction to physicians in obstetrics as well as in pediatrics were offered during the summer of 1936. Other institutes arranged to train public health personnel as well as to give physicians additional training in this field are referred to elsewhere in this report. The aroused public interest in the protection of women during pregnancy is revealed by the decision of the General Federation of Women's Clubs to conduct under competent medical leadership an intensive nation-wide campaign for the reduction of the maternal death rate during 1937.

The close association between unnecessary maternal deaths and the fetal and neonatal death rates is well recognized,[12] and this movement to deal more effectively with the maternal mortality problem should be reflected in a lowered infant mortality rate. In addition to considerations of mortality, protecting and promoting the health of women during pregnancy is an effective means of removing many health hazards from infants.

CHILD HEALTH SERVICES

Previous reviews in this series have dealt extensively with the plans of the Federal Government through the instrumentality of the Social Security Act to promote the work of the individual states and local units of government in behalf of maternal and child health. Funds have now been available for these purposes for more than a year under the terms of that Act, but to only those states which have presented to the Children's Bureau an acceptable program and which have set up a division within the state department of health under the direction of a competent physician to administer maternal and child health activities. According to the latest reports all of the states, as well as the District of Columbia, Hawaii, and Alaska, have met the requirements and are now receiving financial assistance for maternal and child health services. It is anticipated that in the near future all will also have a program in operation for crippled children. Recent developments in the operation of these plans have been reviewed by the Assistant Chief of the Children's Bureau[13] and are in accordance with the policies previously outlined. The following quotation from this report referring to the three parts of Title V of the Act (having to do with maternal and child health, crippled children, and child welfare respectively) reviews briefly the scope and method of operation:

"In all three parts of the title preventive services are stressed. The maternal and child health program is entirely a preventive one; in the crippled children's program, though medical and surgical care and

hospitalization of crippled children are the chief services rendered, nevertheless care of children suffering from conditions that may lead to crippling is specifically authorized in the act, and such preventive services are actually being rendered at this time in a number of states, notably in Mississippi, Alabama, and Tennessee, where the recent epidemic of poliomyelitis has occurred. In the child welfare program attention is being directed toward the prevention and amelioration of those social and economic conditions that lead to maladjustment of the individual child in his relation to family and community. The act permits the state public welfare departments in cooperation with the Children's Bureau to make grants to local committees in order to pay part of the cost of services for dependent, neglected, and homeless children, and children in danger of becoming delinquent, with the purpose of providing for children living in the rural and semirural areas the type of care that has been available for these socially handicapped children in larger cities for many years."

According to budget estimates for 1937 slightly more than one-half of all public funds to be expended for maternal and child health services in the average state will be paid out of federal funds, the remainder being met in approximately equal shares by state and local governments. In the case of expenses for crippled children the state is to provide a larger part, slightly more than the federal government, with the local community contributing only about 15 per cent of the total. Of the funds potentially available to the state from the Children's Bureau, estimates for 1937 indicate that advantage will be taken of about 85 per cent. Further information as to plans and policies and recent developments under the Social Security Program is contained in the latest annual report of the Secretary of Labor,[14] and interesting current news is presented in the newest periodical published by the Children's Bureau, entitled, "The Child."[15]

The extension of venereal disease control in the United States through the provisions of the Social Security Act[16] has direct bearing upon child health because congenital syphilis is recognized to be theoretically preventable but as practically requiring community effort to secure satisfactory results. The provision by the public health department of free drugs to physicians does not constitute an adequate program. Epidemiologic measures applied to the search for sources of infection, more energetic following of lapsed cases, and extension of prenatal care with routine testing and prompt treatment of positive cases are principal features of most of the proposed programs. It has been recommended that a full-time venereal disease control officer be appointed in every state department of health and that advisory committees be appointed by state medical societies to recommend practical local programs to the public health service and to the state department of health. There is considerable diversity of program and amount of progress in the various states, but thus far, adequate programs are in few instances in full operation.

PUBLIC HEALTH LAWS AND PROCEDURE

There has been notable progress in recent years in the formulation of principles of public health as they apply to children and this is being reflected in many quarters in revised public health laws and procedure. There is no uniformity in this respect, however, and many unsound and antiquated laws are still at least theoretically operative and often block-

ing effective action by health departments. In 1931 a special commission, appointed by the Governor of New York State to study administrative and legislative aspects of public health in that state (exclusive of New York City), submitted its report.[17] One section of this report dealt with maternal and infant hygiene and another with school hygiene, while others had direct bearing upon child health problems in such special fields as dental hygiene, mental hygiene, communicable disease control, and rehabilitation of crippled children.

During 1936 a commission, appointed by the Governor of Massachusetts to "study and investigate public health laws and policies in the Commonwealth," completed and published its report.[18] This commission, with the aid of numerous committees appointed to deal with special subjects, attempted a comprehensive analysis of the laws and policies relating to maternal and neonatal hygiene and child hygiene. The report recommends changes in these in many instances where they were found out of harmony with present knowledge. Even though this report was concerned only with conditions in Massachusetts, it constitutes a valuable contribution to the literature on public health principles and practice, especially in helping to clarify fundamental issues. The report is worthy of further study by those interested in public health but the following quotations may be appropriate as suggesting the nature of its content:

Recommendations by the committee dealing with neonatal hygiene:

1. "That health departments and the medical profession attempt to increase the demand for, and quality of, adequate obstetric care.
2. "That the licensing of maternity hospitals be made a function of the Department of Public Health.
3. "That the Department, in conjunction with the medical societies, conduct a detailed study of maternal deaths.
4. "That a special form be used in the recording of stillbirths, and these be handled in the same manner as death certificates.
5. "That detailed study be made of neonatal deaths according to areas and maternity hospitals.
6. "That maternity hospitals be licensed only if they are properly equipped to care for premature infants.
7. "That births of premature infants be made reportable immediately to the local board of health which, in case of home deliveries, should be responsible for necessary transportation to a properly equipped hospital.
8. "That the care of premature infants so transported to a hospital be a public welfare charge without pauperization, if the family is unable to pay."

From the report of the committee on infant and preschool child hygiene:

". . . such educational and preventive health services as are necessary and effective in forestalling serious diseases and handicaps in early life should be available to the entire population. . . . Child health services which should be available, over and above an adequate general public health program, deal with the education of parents and with continuous protective care of infants and children.

". . . The services of a physician are . . . necessary wherever individual health supervision is to be provided. Ideally . . . best given by the physician who prescribed for the child in illness and who is familiar with the family background.

". . . All agencies should cultivate a desire on the part of parents for regular preventive health supervision for their children. The desirability of securing this service from a private doctor should be constantly stressed. This is important not merely from an economic standpoint but because of the greater assurance of securing maximum health protection when medical care in both health and illness is highly personalized, and is combined and continuous under one physician. Effective service can be made available in the physician's office at a fee within the reach of most people, provided . . . it is extensively sought. . . . The community health conference is an extremely useful and proper agency to provide minimum essential service for those unable to pay for these services from a private physician. Such conferences can meet the most glaring and urgent needs for health protection, but must concentrate upon essentials.

". . . Health examinations, immunizations, and advice should be available at the time of a single visit. If a private physician provides one of these services for a particular child, he should provide all.

". . . Community resources for correction of remediable defects must exist if the conference is to be effective."

Other recent publications dealing with public health laws and procedure which deserve especial attention are "Public Health Administration in the United States" by Smillie[19] and "Rural Health Practice" by Mustard.[20]

TRAINING OF PUBLIC HEALTH PERSONNEL

The contribution to the health of a community made by its public health services is in large measure dependent upon the ability and training of those who administer and those who provide these services. Leaders in this field have long been endeavoring to raise the educational requirements for public health office, and in turn the standards of the schools and university departments which give instruction in hygiene and public health.

The first "School of Public Health" was established jointly by Harvard University and the Massachusetts Institute of Technology in 1913, and the second by Johns Hopkins University in 1918. Harvard University established its own independent school in 1922 replacing the organization which had functioned since 1913, and Toronto University opened the third and last School of Public Health on this continent with independent faculty and buildings in 1925. Numerous universities and medical schools have in addition developed departments of hygiene and public health, some of which grant certificates or degrees in public health to graduate students. This movement has been greatly accelerated during the past two years by the passage of the Federal Social Security Act, providing both an increased need for well-trained health officers to fill the many new positions created by the Act and also new sources of funds for certain types of training. The University of California, for example, has for these reasons within the past year established a department of public health prepared to give certificates to graduate students. Among other universities now offering certificates or degrees in public health to graduate physicians might be mentioned Yale, Columbia (through the Delamar Institute of Public Health at the College of Physicians and Surgeons), Vanderbilt, the Universities of North Carolina, Michigan, Minnesota, and Kentucky; the Massachusetts Institute of Technology, and Tulane University.

The graduate schools of public health have greatly increased their enrollment of candidates for degrees and certificates since the adoption of the Social Security Act. For example, the Harvard School had 22 students enrolled in 1934-35 and granted 18 degrees of certificates that spring. In 1935-36 this school had an enrollment of 35 and granted 26 degrees or certificates, and during this academic year there has been an enrollment of 59 students.

In addition to the need for an increased number of well-trained public health officers to fill new positions, the raising of the general level of standards by various health departments throughout the country has created a great demand for short-term, or "refresher," courses for present incumbents of health department positions. Special training facilities have been developed for this purpose at various centers. Closely allied with this has been a movement to offer practicing physicians an opportunity through extra-mural medical education or through short courses at various centers, to learn more about preventive medicine and public health. Until students now being trained, or who have recently been trained in our medical schools, come to take a prominent part in the general practice of medicine throughout the country, chief reliance for increasing the participation of general practitioners in preventive services must be placed upon various forms of postgraduate education and upon introducing these subjects into medical society programs.

In calling attention to the stimulus given to the training of public health personnel as a result of financial assistance from the Federal Government, the continuing part played by the large Foundations which pioneered in this field should not be overlooked. The Rockefeller Foundation, which has contributed more than any other organization toward the development of present training facilities in this country, continues to function primarily through grants to or endowment of the several Schools of Public Health, and by establishing fellowships for prospective health officers in these schools. The Commonwealth Fund[21] has continued to stimulate the teaching of preventive medicine, with special emphasis on obstetrics and pediatrics, in a variety of ways. To date over 400 physicians have been granted postgraduate fellowships, usually of four months' duration, to attend medical schools for "refresher" courses. These physicians have been chosen from states in which the Fund has been financing special public health activities and from the staffs of rural hospitals sponsored by the Fund, so that their training has been part of a broader undertaking, looking toward the improvement of both public health practice and private practice in the field of preventive medicine. In addition to these fellowships, the Commonwealth Fund has contributed to public health education through grants to universities to provide for adequate departments of preventive medicine and for extension courses.

The Calhoun County, Michigan, Health Department in cooperation with the Kellogg Foundation may serve as an example of postgraduate service. This department has been conducting an educational program for its practicing physicians and it reports practically 100 per cent of the county physicians taking part in this program during the past three years. This has included a two weeks' intensive training course, speakers for local medical meetings, and meetings of physicians to discuss local situations regarding the health department program and methods of prevention in the field of public health. This kind of undertaking

should do much to encourage local physicians to participate more actively in services concerned with the health of the community as a whole, and their response has been most encouraging. The University of Michigan has also developed an extensive program of instruction in hygiene, public health, and preventive medicine for various types of students, with rather special emphasis on the preparation of teachers and nurses.

In connection with these expanding facilities for training in the field of public health, emphasis has increasingly been placed upon subjects relating to maternal and child health. Instruction of health officers in these subjects must obviously be somewhat different from that afforded medical students or practicing physicians in both content and point of view, but the science upon which procedure is based is the same for both. It is therefore essential that those who teach in these fields be not only well qualified in their own specialties as physicians but that they be broadly experienced and interested in the application of these specialties to public health procedure. Instruction in maternal and child hygiene is being given more and more by obstetricians and pediatricians who are able to bring a knowledge of sound medical principles and practice to those considering community problems. The Department of Child Hygiene at the Harvard School of Public Health, for example, has recently added an obstetrician to its staff, and he will in the future, on a full-time basis, promote research and teaching in the fields of maternal hygiene and the prevention of obstetrical complications. Medical students and students of public health are also having increasing opportunities to work in maternal and health conferences, and to observe community methods of teaching health and giving preventive service in the field.

At the Harvard School of Public Health, in addition to the course in maternal and child health provided for its regularly enrolled students, a series of special two-month training courses have been in operation during the past year, with the cooperation and assistance of the Departments of Health of the New England States under Federal (Social Security) grants. These courses have provided intensive instruction and close personal contact with health services for small groups of health officers who are specializing in maternal and child health activities. As these special courses have been designed to meet a temporary demand for persons with some training in these fields, requirements as to past training of those admitted to the courses have not been strict. Their future usefulness would seem to depend upon restriction to physicians well grounded in the general fields of public health and with some training in pediatrics. The School of Hygiene at Johns Hopkins University, in cooperation with the Departments of Health of the State of Maryland and the City of Baltimore, has also been increasing the opportunities offered to both health officers and medical students to observe maternal and child health activities in city and state. Various departments of preventive medicine and hygiene at other universities have introduced courses in child hygiene.

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News and Notes

The American committee for the **Fourth International Pediatrics Congress** to be held in Rome, wishes to call attention to the final dates of the congress which have been fixed for September 27-30, 1937.

Tentative plans for the congress have recently been announced by the central committee: The first day is to be devoted to a consideration of mineral and water metabolism in infancy, to be discussed by Dr. James L. Gamble of Boston, Dr. E. Freudenberg of Marburg, Dr. Noah Morris of Glasgow, Dr. Czapo of Budapest, Dr. Gorter of Leyden, Dr. Speransky of Moscow, Dr. von Reuss of Vienna, Dr. Toverud of Oslo, and others.

The second day will be devoted to tuberculosis in infancy. Among those already scheduled to participate in the discussion are Dr. Chester Stewart of Minneapolis, Dr. Dufour of Lyon, Dr. Brinchmann of Oslo, Dr. Capon of Liverpool, Dr. Wallgren of Gothenburg, Dr. Debre and Dr. Armand-de-Lille of Paris, Dr. Parsons of Birmingham, Dr. Noeggerath of Freiburg, Dr. H. Koch and Dr. Nobel of Vienna, and Dr. Jemma of Naples.

The third day will be devoted to a discussion of neuropsychiatric disorders in pediatrics from the clinical and social point of view. Among those already scheduled to participate are Dr. Dingwall-Fordyce of Liverpool, Dr. Glanzmann of Bern, Dr. Jundell of Stockholm, Dr. Frederic H. Allen of Philadelphia, Dr. Babonneix of Paris, Dr. Barbosa of Rio de Janeiro, and Dr. Sychareva of Charkov.

The fourth day will be devoted to communications on miscellaneous topics.

Those interested in public health may wish to remain for the meeting of the International Congress for the Protection of Infancy, which takes place in Rome immediately following the pediatrics congress.

The Italian committee is preparing a program of entertainment for the members of the congress. These international meetings should continue to be, as they have been in the past, a stimulus and a pleasure, a source of new ideas, and an opportunity for making new friendships and for renewing old ones. The American committee is anxious to make the attendance and the papers from this country as representative of American pediatrics as possible. The committee will welcome any suggestions that may be made. The time for application to present papers closed May 15. Dr. L. Emmett Holt, Jr., Johns Hopkins Hospital, Baltimore, Md., is secretary of the American committee.

Book Reviews

American Medicine: Expert Testimony Out of Court. Two volumes, New York, 1937, The American Foundation, 565 Fifth Avenue. Price, $3.50.

From its very nature it is impossible to discuss this important report in detail. It can only be urged that it should be read by every physician. The American Foundation wrote to hundreds of physicians in all parts of the United States, representing every field and division of medicine, asking for their personal views on the perplexing social, economic, and educational problems of medicine which have become the subject of so much controversy in the last few years. The report presents the opinions and thoughts of 2,100 physicians and is based on over 5,000 letters. The American Foundation has no ax to grind, no pet social hobby to propagandize, no fixed social or economic theory to further which supposedly will solve or do away with the problems of health and sickness. It is not "backing" a state or socialized medicine. It planned, and has accomplished, the task of assembling a tremendous cross-section view of what physicians themselves think of the relation of medicine to the political, social, and economic trends and problems of today. It is an honest, unprejudiced, expression of physicians themselves and as such should furnish an important background for guiding the changes which are taking place. If one wants to know what doctors really think and feel, the answer will in large part be found in this report, as it represents the individual opinions and thoughts of a tremendous cross-section, rather than the views of some one group or clique in control of this or that medical organization. One has to read but a few pages to grasp the sincerity of the purpose of the study and the way it has been carried out, and the integrity of the replies and of those who have been responsible for their compilation. The multitude of viewpoints brought out and discussed in the correspondence have been put together in a most amazing way, and every credit must be given to Miss Esther Lape, who was in charge of the study for the Foundation.

As might be expected in a study conducted in this way, a marked divergence of opinion is expressed in regard to many points. It seems to the reviewer, however, that certain very definite trends of thought, upon which physicians as a whole are in agreement, run through the correspondence, and that these might form a basis for action to which the medical profession as a whole would agree.

Referring only briefly to pediatrics, we find some 4 per cent of the contributors are pediatricians. This is a larger percentage than the ratio of pediatricians to physicians as a whole, but not large when the relation of pediatrics to the social problems of medicine is considered. The progress which pediatrics has made in recent years, and the important strides it has made in its position in medical practice and in the formulation of the present-day curriculum of the medical school are pointed out. In a discussion of medical education this is referred to once as exemplifying the result of conscious propaganda on behalf of a limited special field. To a certain extent there is an element of truth in this, but it should be further pointed out that all the propaganda in the world would have been unsuccessful had there not been two fundamental factors involved: first,

preventive medicine has made its greatest strides and accomplishments in the field of pediatrics, and second, what may be termed "child consciousness" has been an outstanding expression of social trends and developments in recent years.

The importance of this study cannot be overestimated. Its value lies in the fact that it is an expression by physicians themselves of their attitudes and thoughts toward medical problems. To some, at first thought, the report may seem confusing. It will undoubtedly be asked why no conclusions are reached and no plan formulated from this mass of material. It lies in just this fact: the book represents and gives expression to viewpoints and thoughts, rather than a pro and con discussion of some specific proposal, and therein its greatest value and importance are found.

B. S. V.

Clinical Psychology by C. M. Louttit, Director, Indiana University Psychologic Clinics, New York, 1936, Harper & Brothers.

The purpose of this book is to organize the widespread materials in the field of applied psychology, called clinical, into a systematic presentation. The material presented is limited to behavior problems of children.

Part 1 deals with methods of examination. In an introductory chapter the author gives a general description of the field which includes the definition and aims of clinical psychology. The term "clinical" is justified since the aim of clinical psychology is to define the behavior, capacity, and characteristics of the individual, and then give suggestions for the proper adjustment of that individual. This necessarily involves diagnosis and treatment, their limitation to the individual, the child or the adolescent, and to patients in whom there is no organic disease. It finally includes the relationship of other professions, education, pediatrics, psychiatry, mental hygiene, and social welfare. It is quite obvious then that such activities cannot be limited to the meaning of the term psychology, although this is regarded by the author as the basic science of this endeavor.

In Chapter 2 the author describes the examination of the individual. He stresses the importance of inquiring into the child's past history, present living conditions, his performance abilities, his repertoire of skills, habits, and his physical condition. The author describes in detail various methods of testing the child's intelligence and mentions many other characteristics which would come under education, achievement, aptitude, personality, conduct, and environment. A very adequate list of references is included in these chapters.

Numerous brief case records are given to illustrate the various points. The author emphasizes the fact that the job of the psychologist is to evaluate the causative factors and suggest means of correcting the causes of the various difficulties. The earlier in the course of the school period that this correction is made, the better.

Treatment of primary behavior problems calls for changing the cultural milieu with the cooperation of the parents, and in the new surroundings carrying on retraining to achieve more acceptable behavior habits. To a limited extent this same program can be applied to indirect primary behavior problems, but in these an effort must be made to help the child overcome the intermediate attitudes or conflicts. The first of the primary behavior problems discussed at some length are those relating to conduct. They are divided into three groups: first, forms of behavior objected to in the family group; second, problems important in connection with the home, but which have wider social significance; third, problems which are of serious social significance since they violate legal requirements and therefore are broadly antisocial. Individual problems under these headings are discussed in detail and the works and opinions of various investigators are given.

In these cases treatment must be carried out by the parents. They must be taught that the importance of a symptom is dependent upon the cause—not the result. Prevention is more important than treatment, so the author gives suggestions of methods of training the child so that difficulties may be prevented. When the problem has arisen, however, the cause of the disorders depends largely upon the reaction of the individual child and parents or guardians, and the parents or guardians should be instructed in means of training the child so as to eliminate the cause. A number of cases are given to illustrate these questions.

Juvenile delinquency is especially considered in this book. A great deal of work has been done on this subject and the findings are quite uniform, so the author gives information which he has obtained mainly from three extensive studies of large groups of delinquents. Most delinquents show a rather low intelligence (from 80 to 90), are apt to be emotionally unstable, are often native-born children of foreign-born parents, live in homes which are economically and morally poor and in which the family relationship is psychologically bad, and in neighborhoods which are frequently socially disorganized and physically deteriorating. While delinquency is the result of inadequate training and is the symptom of personality disturbances, the causative factors are so numerous that the author gives various illustrative cases rather than attempting to discuss them in a general way.

The psychologic treatment of delinquency patients differs little from that in any other behavior problem, but in these cases behavior involves legal offenses and the child is seen only after he has been to court where the first duty of the judge is to protect society. The author discusses in detail the legal procedures in cases of juvenile delinquency and shows that while the law must protect society from the acts of the offender and the psychologist is concerned with the child as an individual, there is no reason why the two views should conflict. He suggests active psychotherapy during institutionalization, and probation rather than institutionalization as punishment. The probation period can be used as a means to watch the child and to help him to reform, but if his behavior continues to be bad he can then be sent to an institution. The author recommends the cooperation of court and psychologic clinic.

There is a chapter on speech defects. A great deal is said about fears, anxieties, jealousy, and daydreaming. There is also a very intelligent comment on psychoses, particularly dementia precox, schizophrenia, manic-depressive psychoses, and psychoses with organic pathology. Only juvenile paresis is discussed by the author.

It can be seen from this brief sketch of the contents that the book represents a very broadly outlined consideration of the whole subject. A vast amount of material is presented in a systematic form. The student of clinical psychology, for whom it is written, can find here a complete description of his whole field of work as well as references which will enable him to make a detailed study of any subject in which he is particularly interested. The author gives first a general survey of the subject, including examinations, and then devotes the remainder of the book to problems with which this branch of science deals. Throughout he emphasizes the fact that the book deals with the individual, not the problem. He also stresses the fact that the problems are not clear-cut, but merge one into the other. He gives the varying theories of investigators and often gives his own opinion as to which seems most adaptable. In no case does he quote theory as a fact. While the author seems to give more space than is necessary to conditions which are relatively rare, or to those which are the primary concern of others, such as disabilities in school subjects and problems correlated with physical defects, the reader realizes on further consideration that he gives the student such a clear idea of these that he will not be forced to look farther. This

means of presentation serves also to bring out his second important point—that cooperation between this and the other related professions is to be most earnestly sought.

This outline gives but a meager description of the scope and plan of this book. It is by far the most useful book on clinical psychology that the present reviewer knows. It seems to contain all that is essential for the clinical psychologist as well as the psychiatrist or pediatrician with whom he works. The reviewer is impressed with the care and conscientiousness of the author in his task, the lack of prejudice, or any indication of a limited or narrow point of view. The point is constantly stressed that the important thing is the consideration of the child as a whole, and the problems that the child presents are to be considered from the standpoint of the total personality in relation to his total environment. This book is an extensive performance, well over 600 pages in length. It is, therefore, in a sense, a book of reference and this the reviewer thinks is perhaps to be its most important service. The book is clearly written, well arranged, and carries out the plan as presented by the author in his introductory chapter.

S. I. S.
M. L.

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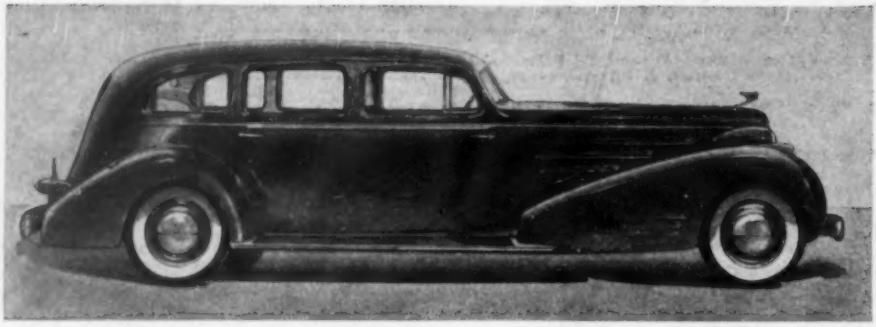

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